STUDY OF CLINICAL, BIOCHEMICAL AND ELECTROCARDIOGRAPHIC ASPECTS OF YELLOW OLEANDER POISONING

Dissertation submitted in partial fulfillment of regulation for the award of M.D. Degree in General Medicine (Branch I)

The Tamilnadu
Dr. M.G.R. Medical University
Chennai
March 2009

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certificate

This is to certify that the dissertation entitiled ***"A study of clinical, biochemical and electrocardiographic aspects of Yellow Oleander poisoning"****, herewith submitted by* ***Dr ABHISHEK S****, post graduate in General Medicine Coimbatore Medical College Hospital is the record of a bonafide research work carried out by him under our guidance and supervision from January 2007 to August 2008.*

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF CLINICAL, BIOCHEMICAL AND ELECROCARDIOGRAPHIC ASPECTS OF YELLOW OLEANDER POISONING "** was done by me from January 2007 to August 2008 under the guidance and supervision of **Professor Dr. M. RAMASWAMY.MD.**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MD Degree in General Medicine (Branch I).

Dr. ABHISHEK. S

Place : Coimbatore

Date : 01.12.2008

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Lastly I thank all the **Patients** who co-operated in this study.

Contents

INTRODUCTION

The yellow oleander (Thevetia peruviana) is an evergreen shrub or a small tree in the dogbane family Apocyananceae family that is widely distributed to a broad area from Morocco and Portugal eastward through the Mediterranean region and southern Asia to southern parts of China. Their beautiful yellow flowers and prolific growth makes them popular shrubs for landscaping.

Oleander is one of the most poisonous plants and contains numerous toxic compounds. All parts of the plant contain cardiac glycosides. The major toxic effects are similar to that of digoxin overdose. The pathophysiology includes direct glycoside inhibition of sodium-potassium pump of the heart and increased vagotonia. Symptoms include vomiting, diarrhea, dizziness, bradycardia, sinus and AV nodal block and other cardiac dysrhythmias[2,3] Fatal, DC shock resistant ventricular fibrillation or refractory cardiogenic shock may occur in severely poisoned patients[4].

Accidental ingestion occurs in children due to beautiful yellow flowers and the conspicuous green fruit[5]. Accidental poisonings have been reported form across the world, for example the solomon Islands, Brazil and Australia. However, intentional poisoning in these regions is very uncommon[6]. But the use of seeds / fruits of yellow oleander as a method of suicide is common only in Srilanka and South India. Since 1980 there has been a sudden increase in the number of cases of suicidal yellow oleander poisoning in Srilanka with thousands of cases occurring each year now with a case

fatality rate of at least 10%[4].

Though the poisoning is a common method of suicide in South India there has been no published reports regarding this poisoning from south India. Data regarding incidence and case fatality rate of this poisoning in south Inidan population is not available. The only Indian study involving large number of patients is from Eastern India. (300 cases, Bose TK et al, 1999)[7]. Hyperkalemia which has been noted in about 30% of the cases of yellow oleander poisoning in a Srilankan study has not been described in Indian studies involving 300 patients and 32 patients respectively [7,8]. The risk factors for cardiotoxicity and outcome have also not been studied in detail in previous studies. Moreover further studies are required to support the use multiple doses of activated charcoal and antidigoxin Fab fragments in yellow oleander poisoning.There has been only one clinical trial each to support their use in yellow oleander poisoning [9,10].

So this study was undertaken to find out the incidence of yellow oleander poisoning among admissions in general medicine wards of our hospital, to study the socio-clinical aspects, to find out correlation between clinical and biochemical parameters with electrocardiographic changes and to assess possible risk factors for cardiotoxicity and outcome and to compare the results with previous published reports.

AIMS AND OBJECTIVES

1. To find out the incidence of yellow oleander poisoning.
2. To analyse the clinico-social aspects.
3. To correlate clinical and biochemical data with electrocardiographic changes.
4. To identify the possible risk factors for cardio toxicity and outcome.

REVIEW OF LITERATURE

Yellow Oleander (Thevetia peruviana), - Plant description and distribution.

It is a small ornamental tree belonging to family Apocynaceae which grows to about 10 to 15 feet high. The leaves are in pairs or whorls of three, thick and leathery, dark green, narrow lanceolate, 5-21 cm long and 1-3.5 cm broad, and with an entire margin. Flowers are bright yellow and funnel shaped with 5 petals spirally twisted. The flowers grow in clusters at the end of each branch; bright yellow, 2.5-5 cm diameter, with a deeply 5-lobed corolla with a fringe round the central corolla tube, which are often, but not always, sweetly scented. The fruits are somewhat globular, slightly fleshy and have a diameter of 4 to 5cm. The fruits which are green in colour, become black on ripening. Each fruit contains a single nut; light brown in colour and triangular in shape with two cells, each enclosing a pale yellow seed[5].

These plants are widely distributed throughout the tropical to the sub temperate zones of the world, typically occurs around dry stream beds. Their beautiful yellow flowers and prolific growth makes them popular shrubs for landscaping. These plants also may be found growing wild[1]. It is often planted in numbers as a hedge[11].

Yellow oleander tree (Thevetia peruviana)

Yellow oleander flower

Historical and Medicolegal aspects

Many myths are associated with this plant. In the West Indies, the nut is carried in the pocket in the belief that it will ward off hemorrhoids. In East Africa, it is put in the hand of an infant at birth as a good luck token. In Africa, the seed kernels are occasionally chewed to cause purging. In the Philippines, half of one leaf in given as emetic and purgative. The sap and bark have been utilized in small amounts to treat malarial fever as well as to induce vomiting and purging. The sap also has been applied to sores and ulcers, also to tooth cavities and decayed teeth to relieve toothache[11].

The toxicity of the plant has been generally known since the sixteenth century. It's Sanskrit name "Ashwamarak" is translated as horse killer[11]. Accidental poisoning occurs in children as they may play with and taste the bright yellow flowers and conspicuous green fruit. The seeds are used for suicide attempts particularly by young people especially in northern parts of Srilanka and South India. Other medico legal aspects include use of roots and seeds with water or oil for procuring criminal abortion and seeds for poisoning cattle[5]. Yellow oleander glycosides proved effective in patients with heart failure and atrial fibrillation in studies carried out in 1930s, however digoxin has been preferred because of less frequent gastrointestinal side effects[12].

Epidemiology of yellow oleander poisoning

Ingestion of oleander seeds or leaves is a cause of accidental poisoning worldwide, particularly among children [6,13]. Cases have been reported from places as

diverse as Hawaii, the Solomon Islands, Southern Africa, Australia, Europe, the Far East and the United States[14,15]. However the use of seeds / fruits of yellow oleander as a method of suicide is common only in Srilanka and South India. This method of suicide is common among adolescents and young adults with a female preponderance [4,7]. Currently, thousands of cases of yellow oleander poisoning occur in Srilanka every year with a case fatality rate of at least 10%[4]. The exact incidence of the disease in Indian population is not known. Bose TK et al (1999) noted a case fatality rate of 4.6% among 300 cases in Eastern India[7].

Poisonous parts

Generally, pulp of the seed is crushed into a paste for this purpose. All parts of the plant are poisonous especially the seeds / kernels of fruit[16].

Poisonous agents

In the decreasing order of potency the following cardiac glycosides - Peruvoside, Ruvoside, Thevetin A , Nerifolin, Gerebrin and Thevetin B[17] are the poisonous agents found in this plant.

Pathophysiology of toxicity

The poisonous agents found in yellow oleander plant are cardiac glycosides. These bind to a site on the cell membrane, producing reversible inhibition of sodium (Na^+) - potassium (K^+) - adenosine triphosphatase (ATPase) pump, which causes

increased intracellular sodium concentrations and decreased intracellular potassium. In myocytes, elevated intracellular sodium concentrations produce increased intracellular calcium concentrations via a sodium (Na^+) – calcium (Ca^{2+}) exchanger. Excessive intracellular calcium is concentrated in the sarcoplasmic reticulum and released in excess, with depolarization[18].

Release of excessive calcium results in enhanced cardiac contractions, which are delayed after depolarization and manifest clinically as after contractions, such as premature ventricular contractions. Cardiac glycosides also have vagotonic effects, resulting in bradycardia and heart blocks. Inhibition of Na^+-K^+-ATPase in skeletal muscle results in increased extra cellular potassium and contributes to hyperkalaemia[18]. Severe hyperkalemia can contribute to atrioventricular (AV) block and depressed myocardial excitablity[19].

Cardiac glycosides primarily affect cardiovascular and gastrointestinal systems, of which effects on the cardiac system are most significant. The pathophysiology that produces cardiotoxicity involves prolonging refractory period in the atrioventricular (AV) node, shortening refractory periods in the atria and ventricles, and decreasing resting membrane potential (increased excitability). Any dysrhythmia characterized by both increased automat city and depressed conduction is suggestive of cardiac glycoside toxicity[18].

Dysrhythmias often associated with cardiac glycoside toxicity include bradydysrhythmias, sinus bradycardia with all types of atrioventricular (AV) nodal block, junctional rhythms and sinus arrest. Dysrhythmias characterized by increased automaticity and conduction blockade, are highly suggestive of cardiac glycoside toxicity. These include tachydysrhythmias such as atrial tachycardia with block, paroxysmal atrial tachycardia with block, bidirectional ventricular tachycardia and ventricular fibrillation[18].

Thevetin is easily absorbed from the gastrointestinal tract. Thevetin glycosides occur in higher concentrations in heart muscle than in blood[20]. Thevetin probably has a shorter half-life than digoxin and a lower risk of accumulation in the body[17].

Fatal dose

The kernals of the seed are most toxic with the reported lethal dose being 8-10 seeds [5,29]. Recent studies in Srilanka involving larger number of patients found that there was no exact relationship between number of seeds ingested and outcome[4].

Clinical features of yellow oleander poisoning

Symptoms

Closely resembles digitalis poisoning with gastrointestinal and cardiac symptoms.

Gastrointestinal symptoms

Symptoms are nonspecific and include nausea, vomiting, diarrhea and abdominal pain [2,3].

Cardiac symptoms

Include palpitations, shortness of breath, giddiness etc[18].

Neurological Symptoms

Pupils may be dilated[21]. and excessive salivation has been reported[20]. Paraesthesias and weakness have also been reported[20]. Symptoms are often nonspecific and include giddiness, numbness (especially of tongue and oral mucous membranes), altered mental status (eg. Disorientation, confusion, drowsiness, lethargy etc) and occasionally seizures[18].

Complications

1. Cardiogenic shock can develop secondary due to arrhythmias. The various arrhythmias that are noted in yellow oleander poisoning are sinus bradycardia, sino-atrial block, all types of atrioventricular (AV) block including Mobitz type II second degree AV Block, junctional rhythm, supraventricular tachycardia, atrial fibrillation, artrial flutter, bundle branch block, ventricular ectopics, ventricular tachycardia and ventricular fibrillation[8].

2. Hyperkalemia is seen in severe cases of yellow oleander poisoning[8].

3. Acute renal failure can develop secondary to cardiogenic shock.

4. Acid base disturbance-circulatory collapse can cause metabolic acidosis.

Postmortem Appearance

Subendocardial and perivascular hemorrhage with focal myocardial edema has been noted[7]. Generalised hemorrhages and signs of gastrointestinal irritation have also been found[5].

Course and prognosis

In severe poisoning diarrhea, vomiting, abdominal pain and sinus bradycardia are early features. Hyperkalemia, conduction block and ventricular ectopics indicate serious toxicity[20]. Indicators of a poor prognosis include multiple and varying cardiac rhythms, with sino-atrial and atrioventicular blocks in combination with ventricular excitability, ST depression over 2.5mm and unresponsiveness to atropine[2]. Conduction block and sinus bradycardia may persist for 5 days after ingestion. Patients usually recover from these if no underlying cardiovascular pathology exist[20].

Management

Investigations

Blood glucose estimation assess for hypoglycemia as a possible cause of altered mental status, electrolyte disturbances. Management is very similar to that of digoxin

poisoning.

Decontamination

If consciousness is not impaired, emesis can be induced or gastric lavage can be performed. Gastric lavage is most useful when started within sixty minutes after ingestion[24].

Activated Charcoal

Activated charcoal can be given 60-100g orally or via gastric tube mixed in aqueous slurry[24]. Multiple doses of activated charcoal (50g of activated charcoal every 6 hrs for 3 days) has been found to be more effective in reducing deaths and life-threatening cardiac arrhythmias after yellow oleander poisoning than single dose. After absorption into the systemic circulation cardiac glycosides are secreted into the gut lumen by the action of p-glycoprotein (enterohepatic circulation). In gut, activated charcoal binds the secreted glycoside and encourages further secretion, thereby causing a rise in glycoside excretion. Through the interruption of the enterohepatic circulation of the cardiac glycosides in yellow oleander, multiple doses of activated charcoal administration improves outcome in yellow oleander poisoning[9].

Electrolytes

Hyperkalemia occurs in severe yellow oleander poisoning. The degree of hyperkalemia correlated with the serum digoxin cross reactive cardiac glycoside concentration[8]. Severe hyperkalemia can contribute to atrioventricular (AV) block and depressed myocardial excitablility[19]. Hypokalemia can also exacerbate cardiac glycoside toxicity due to enhanced binding of cardiac glycoside to Na^{+}-K^{+} ATP ase pump[22]. Electrolytes should be monitored frequently, particularly serum potassium levels. Hypercalcemia and hypomagnesmia also can exacerbate cardiac glycoside toxicity.

Blood urea and Creatinine.

Acute renal failure can develop secondary to cardiogenic shock. Renal insufficiency is associated with elevated endogenous digoxin like immunoreactive factors that can give false – positive digoxin assay results[18]. Renal function has to be closely monitored.

Cardiac glycoside level.

Some plant glycosides cross react with commonly used digoxin radioimmunoassay and digoxin fluorescence polarization immunoassays[23]. Detectable levels of cardiac glycosides have been associated with ingestion of foxglove and oleander ; however levels do not correlate with severity of illness, Beyond its qualitative usefulness in oleander toxicity, however the digoxin serum levels clinical significance is unknown. Negative digoxin radioimmunoassay does not rule out a plant glycoside

exposure[18]. In one of the Srilankan studies there was a correlation between degree of hyperkalemia and the serum digoxin cross reactive cardiac glycoside concentration. But they could not identify cardiac glycoside levels or hyperkalemia at presentation as determinants of mortality[8].

Electrocardiogram (ECG) and continuous cardiac monitoring

To find out the cardiac rhythm, identify life threatening arrhythmias and for treatment.

Hemoglobin level – To determine if anemia is a cause or potential complicating factor for dysrhythmia or hypotension.

Arterial blood gas analysis – To identify metabolic acidosis which may develop secondary to circulatory collapse.

Sample collection – Remaining parts of the ingested plant and gastric contents are useful for botanical identification. Plant portions found in vomitus should be stored in a plastic bag for forensic examination.

Treatment

General principles include providing general supportive care, immediate gastric decontamination, preventing further exposure and absorption, administering antidote and correction of arrhythmias and electrolyte imbalances.

Antidote

Anti-digoxin Fab fragments are a safe and effective treatment for serious cardiac arrhythmias induced, by yellow oleander[10]. The investigators used 1200mg of antidigoxin Fab fragment in a randomized controlled trial involving 66 patients. Flanagan RJ and Jones AL recommend that the approximate dose of Fab fragments (mg) is 80 times the digoxin body burden (mg). They recommend a dose of 380mg of anti-digoxin Fab fragment in an adult if neither the dose for elderly patients nor those with renal impairment should be similar to that for those with normal renal function. The antibody fragments are given intravenously over 15-30 minutes after dilution to at least 250ml with 0.9% (w/v) sodium chloride. Fab fragments are generally well tolerated. Adverse effects include hypokalemia and exacerbation of congestive cardiac failure ; renal function could be impaired in some patients[25]. Indications of use include hyperkalemia (>5.0meq/1), life-threatening supraventricular and ventricular dysrhythmias and hemodynamically significant bradycardia unresponsive to atropine[18].

Treatment of Arrhythmias

Since onset of action of Fab fragments may take 30-60min, intervening treatment of significant complications should be done.

Bradyarrhythmias

Atropine and cardiac pacing can be tried. Patients requiring transcutaneous cardiac pacing should receive Fab fragments prior to it. Transvenous pacing and use of

isoproterenol can result in degeneration of cardiac rhythm and should be avoided. Overdrive pacing should not be used for the control of ventricular dysrhythmias[18].

Tachyarrhythmias

Phenytoin and lidocaine are agents of choice. Magnesium has been reported to reverse digoxin induced dysrhythmias and may be useful as long as anuric renal failure is not present. Quinidine and procainamide may enhance cardiac glycoside toxicity by slowing conduction across AV node ; both should be avoided[18].

Cardioversion is used as a last resort, as it may induce intractable ventricular fibrillation. Fab fragments should be given with cardioversion. If time permits, cardioverison should be attempted after a loading dose of phenytoin and at a significantly reduced initial power setting of 5-10J[18].

Hyperkalemia Glucose, insulin, sodium bicarbonate and salbutamol may be used to facilitate redistribution of potassium intracellularly. However, salbutamol may precipitate cardiac dysrhythmias. Life threatening hyperkalemia should be treated with Fab fragments. Calcium should be avoided in hyperkalemia due to cardiac glycoside toxicity as already there is excess of calcium intracellularly and results in overloading of myocytes with calcium, increased dysrhythmias, and a higher rate of death[18].

Cardiac arrest

Give 10-20 vials of Fab and treat with standard advanced cardiac life support (ACLS) principles. Prolonged efforts at resuscitation may be warranted until Fab fragments begin to work[18].

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Vital signs and biochemical parameters

The range, mean and standard deviation values of pulse rate, blood pressure and biochemical parameters are given in the table below. The mean values of all these parameters were within normal limits. Pulse rate was irregular at admission in 16.2% of the cases, in the rest the pulse rate was regular. Hypotension was present in two cases at admission.

Table No.13 Vital signs and biochemical parameters

Parameter	Range	Mean	S.D
Pulse rate at admission	36-140	80.07	20.9
Systolic blood pressure (mmHg)	70-150	111.8	18.63
Diastolic blood pressure (mmHg)	40-100	73.75	10.57
Blood urea (mg/dl)	15-72	27.19	10.33
Blood sugar (mg/dl)	56-235	91.54	31.77
Serum Creatinine (mg/dl)	0.6-1.8	0.9	0.32
Na^+ (meq/l)	122-160	138.44	5.23
Cl^- (meq/l)	90-106	97.69	2.83
HCO_3^- (meq/l)	15-24	19.33	1.84

were vomiting in 46.9% of cases, numbness sensation in 43.8, dryness of mouth in 25%, diarrhea in 18.8% of cases. Routine blood and urine examination were within normal limits. There was no biochemical abnormalities detected. The ECG change noted were sinus bradycardia in (53%), 1 AV block in 5 (15.6%), III AV block in 1 (3.1%), ST elevation in 2 (6.2%), ST depression in 4 (12.5%) and T wave changes in 6 (18.8% of cases).

MATERIALS AND METHODS

Setting

Department of Medicine, Coimbatore Medical College Hospital, Coimbatore. There was no other collaborating department.

Design of Study

Prospective study

Period of study

January 2007 to August 2008

Sample size

Hundred and six cases of yellow oleander poisoning that satisfied the inclusion and exclusion criteria.

Selection of study subjects

Yellow oleander poisoning cases who fulfilled the inclusion and exclusion criteria.

Inclusion criteria

Those admitted in general medicine wards with history of yellow oleander ingestion during the period of January 2007 to August 2008.

Exclusion criteria

1. Paediatric cases were excluded (<13yrs of age)
2. Those with underlying severe cardiac, renal or hepatic disease were excluded.
3. Patients who were taking the following drugs were excluded – Digoxin , Diuretics , Verapamil , diltiazem , Beta blockers, ACE inhibitors, Amiodarone, calcium and potassium supplements were excluded.

Methods

Selected sociodemographic, clinical, biochemical, electrocardiographic and treatment details were collected from the patients and recorded in a proforma.

Socio demographic data consisted of

- Age
- Sex
- Locality
- Occupation
- Income

Data regarding poisoning comprised of part ingested, quantity of poison, method of ingestion, whether consumption in empty stomach or after food, the intention behind

poisoning, time of ingestion, first aid at home, consumption to admission interval, treatment given, duration of hospital stay and the type of outcome.

Clinical data comprised of

Symptom analysis, pulse rate, rhythm, blood pressure and systemic examination.

Laboratory data included

- Blood sugar
- Blood Urea
- Serum Creatinine
- Serum Na^+, K^+, values
- Electrocardiogram (ECG)

Blood urea, sugar, serum creatinine and serum Na^+, K^+, values were estimated using ERBA XL 300 automated analyzer. The blood urea, sugar, creatinine and serum Na^+, K^+, were measured at the time of admission before instituting treatment. 12 lead ECG, including rhythm strip in lead 11 and V_1 was taken in all patients. 12 lead ECG including rhythm strip was taken at admission before instituting treatment and repeated depending on the clinical status.

Ethical committee approval

The present project was approved by the ethical committee.

Consent

An informed consent was obtained from all patients who were included in the study.

Limitations

1. Continuous ECG monitoring and serial levels of electrolytes like serum potassium and sodium could not be done due to technical constraints.
2. Estimation of cardiac glycoside level was not done due to nonavailability and financial constraints.
3. Activated charcoal, anti-digoxin Fab fragments were not administered and temporary pacemaker not used due to financial and technical constraints.
4. Arterial blood gas analysis was not done due to nonavailability and financial constraints.

Conflicts of interest

There was no conflict of interest.

Financial support

Nil

Statistical analysis

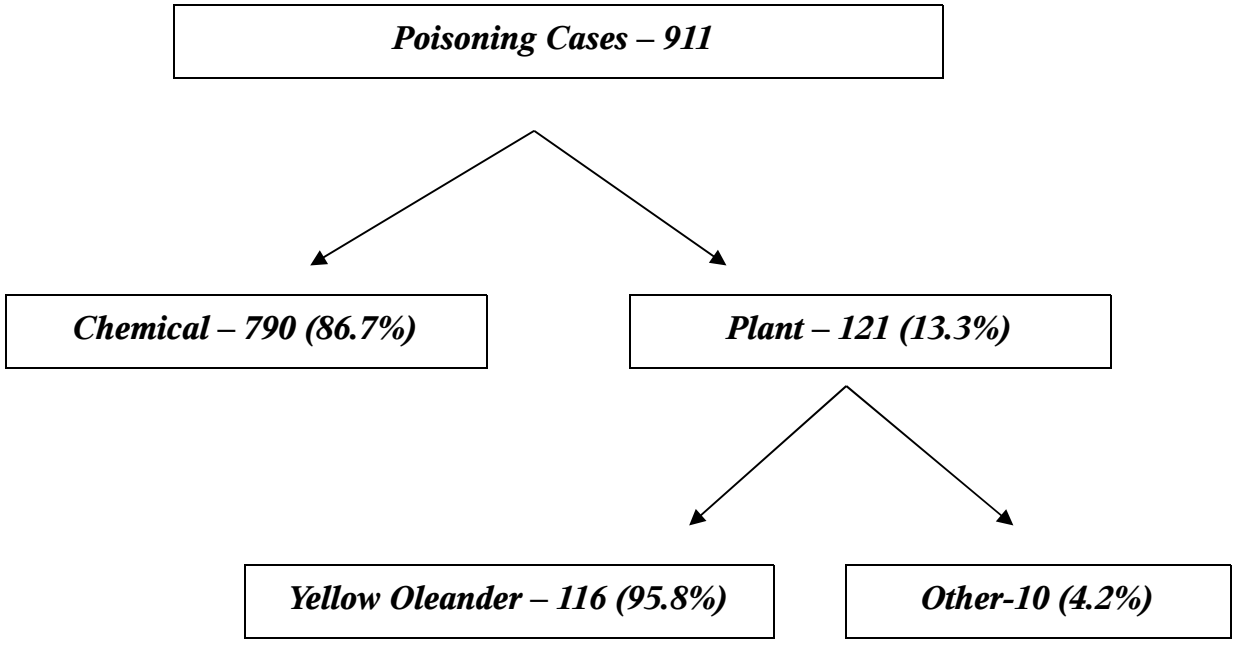
Data were entered in Microsoft Excel spreadsheet and analyzed utilizing the software – Epidemiological Information Package 2002 (Epi Info 2002) – developed by the Centre for Disease Control and Prevention, Atlanta for World Health Organization, Frequencies, percentages, range, mean, standard deviation and ‘p’ values were calculated using this package.

Chi Square test was done to find out the significance of relationship between the groups. SPSS software was used for analysis. The difference was considered to be significant if the ‘p’ value was less than 0.05.

RESULTS

Incidence of Yellow Oleander Poisoning

The incidence of yellow oleander poisoning among total number of admissions in general medicine wards of Coimbatore Medical college Hospital, Coimbatore, during the study period was 11.8 per1000admissions. It accounted for 95.8% of the cases of plant poisoning. In general, 10% of the cases among the total number of admission in general medicine wards were poisoning cases. Among the poisoning cases, 86.7% were chemical poisoning and 13.3% plant poisoning. Among the total number of poisoning cases, yellow oleander accounted for 12.7% of the cases. This is explained using a flow chart.



Distribution of cases in relation to age.

The maximum number of cases occurred in the age group 20-29 (42.5%) followed by age group 13-19 (30.2%) and 30-39 (17%) respectively. The age of the patients ranged from 13-62 years. The mean age and standard deviation was 27± 10. The differences in the mean age of males and females were not statistically significant. The group gives the distribution of cases according to age group and gender. (Figure 1)

Table no: 1 Age group distribution

Age (in years)	Sex of the patient	
	males	females
	percentage	percentage
<20	12.3%	17.9%
20-29		
30-39	18.9%	23.6%
>39	10.4%	6.6%
	4.7%	5.7%

Distribution of cases in relation to gender

Among the total of 106 cases, 49 (46.2%) were males and 57 (53.8%) were females (Figure 2). The ratio of females: males were 1.16:1, but the difference was not statistically significant (P > 0.1).

Table no: 2 Distribution with regard to gender

Sex of the patient	count	percentage

male	49	46.2%
female	57	53.8%

Distribution of cases in relation to income

77.3% of the patients had income below Rupees two thousand per month. The mean income was Rupees 1603 ± 3315 range being Rs. 200 – 30,000. The details are given in the table below.

Table No.3 Distribution of cases in relation to income

Income (in rupees)	counts	percentage
<1000	28	26.4%
1000-1999	23	21.7%
2000-2999	10	9.4%
3000-3999	5	4.7%
>4000	9	8.5%
No income	31	29.2%

Intention behind poisoning

The intention behind poisoning was suicidal in majority of cases (81.1%) and accidental in 1.9% (figure 4). In 17% of cases the poisoning attempt was done just to frighten others for some personal gain or to resolve conflict. There were no cases of homicidal poisoning.

Table No.4 Intention behind poisoning

Intention	counts	percentage
suicidal	86	81.1%
accidental	2	1.9%
homicidal	0	0%
others	18	17.0%

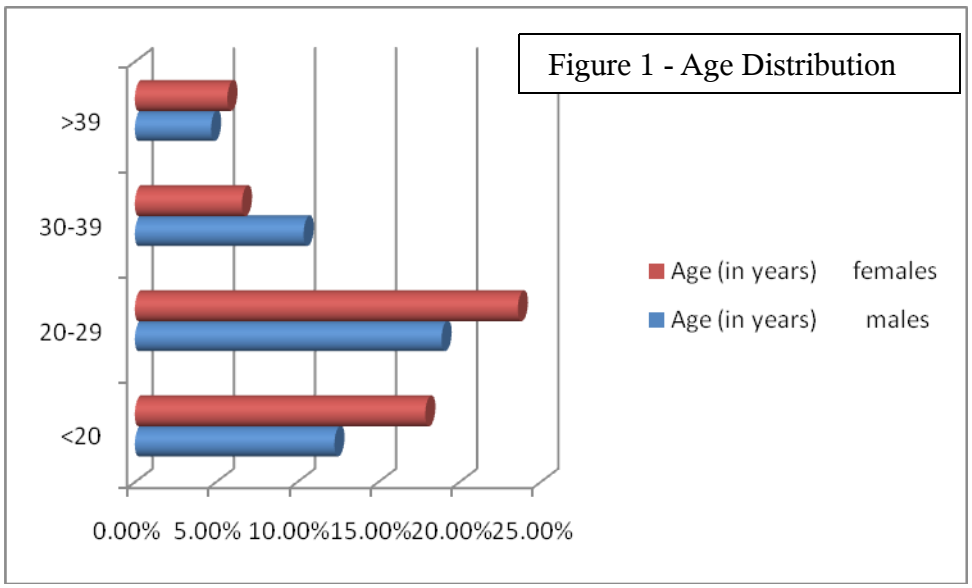


Figure 1 - Age Distribution
>39
30-39
20-29
<20
0.00% 5.00% 10.00%15.00%20.00%25.00%
Age (in years) females
Age (in years) males

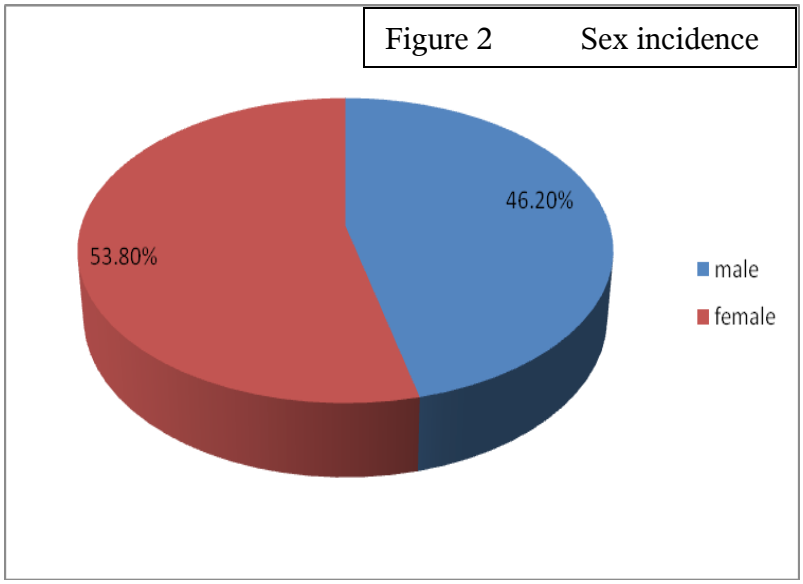


Figure 2 Sex incidence
46.20%
53.80%
male
female

Figure 3 - Income (in Rupees)

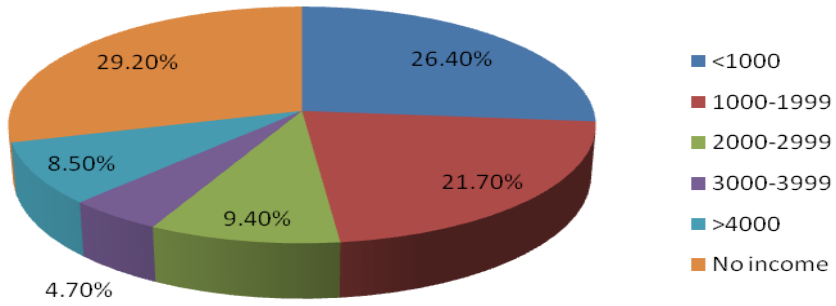
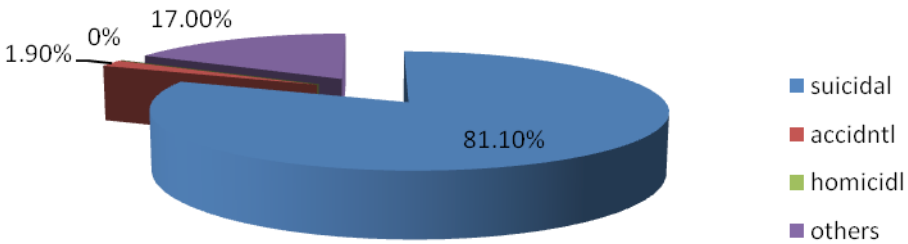


Figure 4 - Intention behind poisoning



Time of ingestion

In majority of cases (84.9%) the poisoning between 6 am - 6 pm. (figure – 5)

Table no.4 Time of ingestion

Time in hours	Persons consumed	percentage
6am- 6pm	90	84.9%

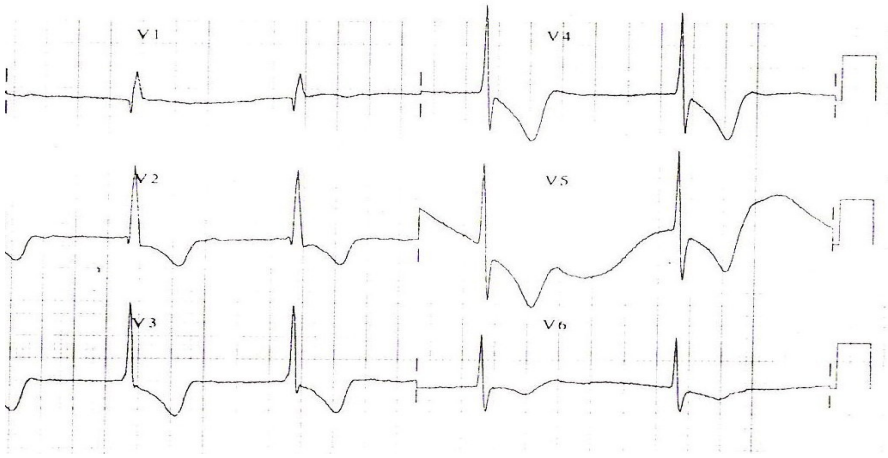
After 6pm	16	15.1%

Electrocardiographic manifestations of yellow oleander poisoning.

The most common abnormal findings were sinus bradycardia (34%) and ST – T changes (40.6%) (Which were similar to that described for digoxin effect / toxicity). Third – degree AV block and first – degree AV Block were noted in 7 cases each. Mobitz type – II second degree AV block which is not described in digitalis toxicity occurred in two cases. Depending on the ECG changes, all cases of yellow oleander poisoning were divided into **no, less severe and severe cardiotoxicity groups**(No cardiotoxicity includes those presenting with sinus rhythm and sinus tachycardia, less severe cardiotoxicity includes those with sinus bradycardia, atrial ectopics,ST-T changes,first degree heart block. Severe toxicity includes junctional rhythm, second degree heart block, third degree heart block and AV dissociation).Figure 8.

Table No.5 ECG changes in yellow oleander poisoning

	ECG manifestations	count	percentage
No cardiotoxicity	sinus rhythm	64	60.4%
	sinus tachycardia	12	11.3%
Less severe cardiotoxicity	sinus bradycardia	36	34.0%
	atrial ectopics	1	0.9%
	first degree heart block	7	6.6%
	ST- T changes	43	40.6%
Severe cardiotoxicity	junctional rhythm	3	2.8%
	second degree heart block	2	1.9%
	AV dissociation	2	1.9%
	third degree heart block	7	6.6%



Effect similar to Digitalis

Figure - 5 Time of ingestion

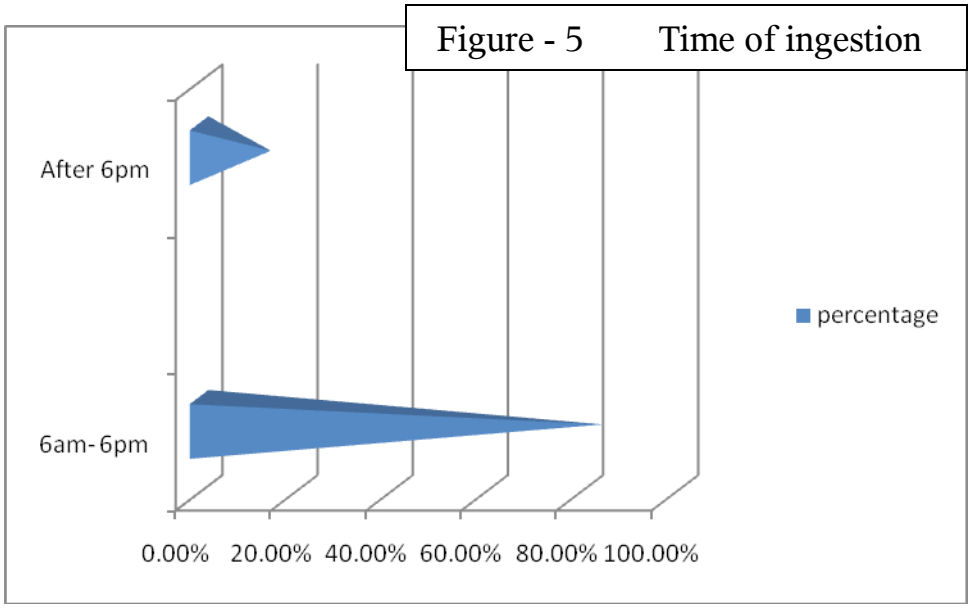
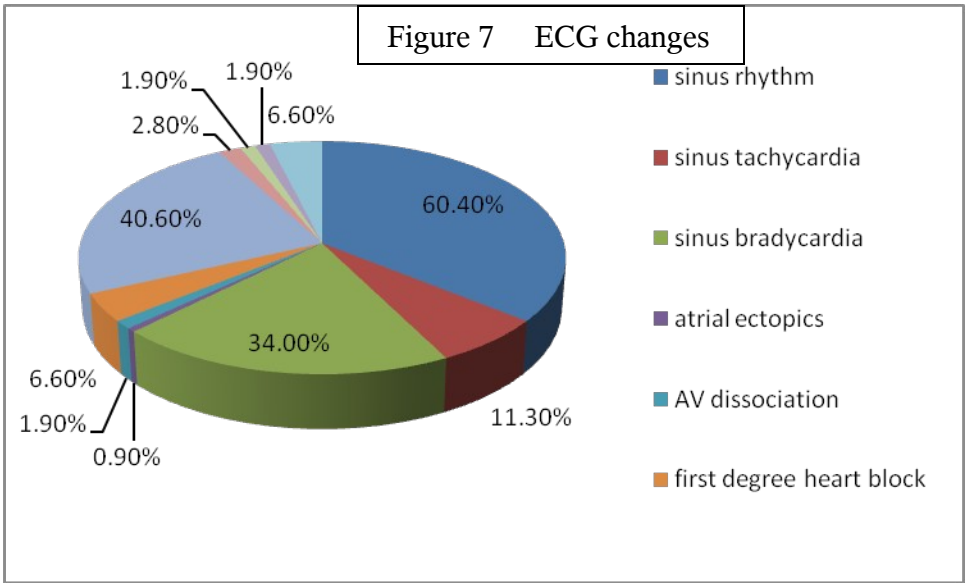
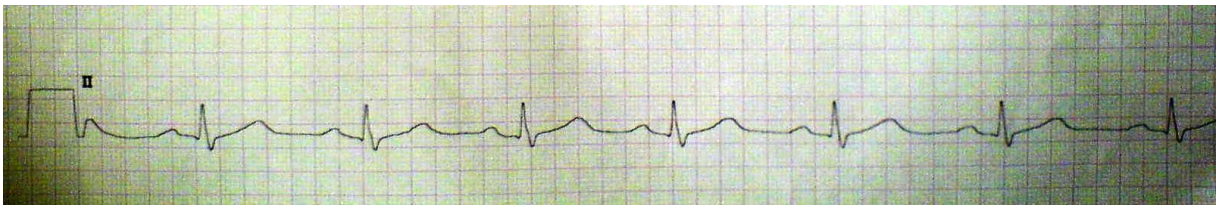
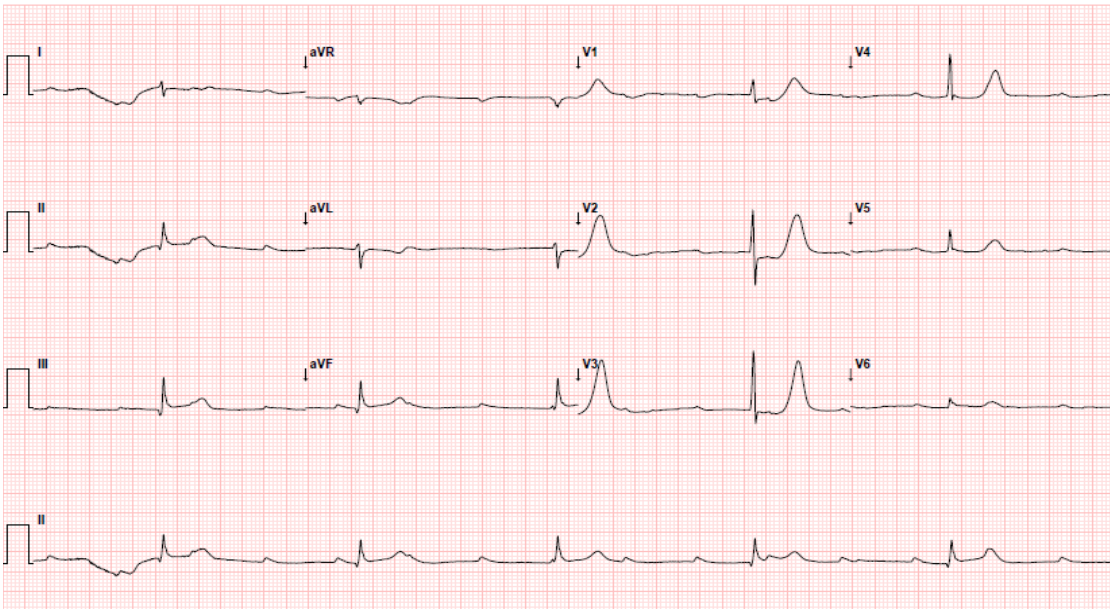


Figure 7 ECG changes





First Degree Heart Block

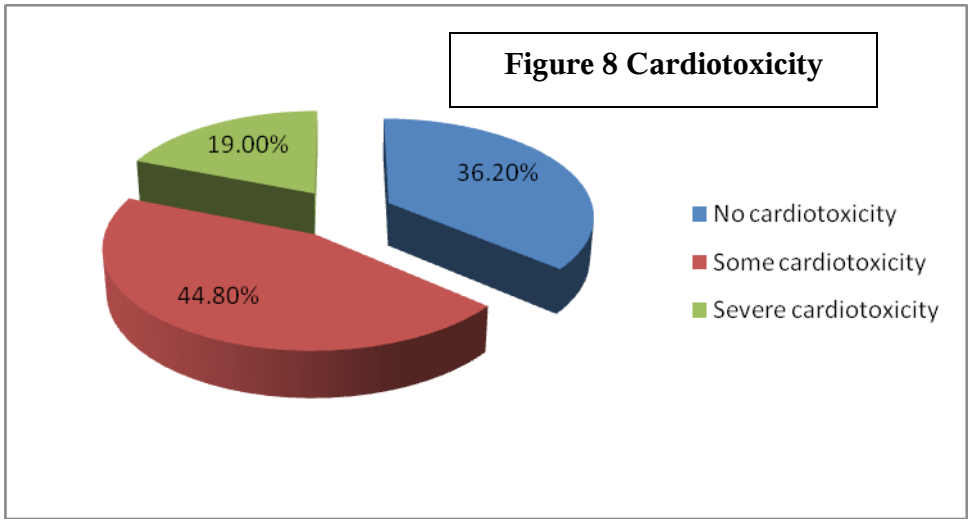


AV dissociation

In majority of the cases (44.8%) some form of the cardiotoxicity was present. Severe cardiotoxicity was present in about 19% of the cases. The details are depicted below in the table and the pie diagram(figure- 8).

Table no.6 Cardiotoxicity

Cardiotoxicity	count	Percentage
No cardiotoxicity	38	36.2%
Less severe cardiotoxicity	47	44.8%
Severe cardiotoxicity	20	19.0%



Relationship between part ingested and cardiotoxicity

Majority of the patients had taken either the fruit or seed, 55 and 51 patients respectively . There was no statistically significant difference in the cardiotoxicity caused by fruit and seed (p>.05). The details are given in the table given below and figure 9.

Table No. 7 Part ingested and Cardiotoxicity

Parts of plant	cardiotoxicity					
	No cardiotoxicity		Less severe cardiotoxicity		Severe cardiotoxicity	
	Count	%	Count	%	Count	%
fruit	19	34.5%	25	45.5%	11	20.0%
seed	19	38.0%	22	44.0%	9	18.0%
Flower	-	-	-	-	-	-
leaves	-	-	-	-	-	-
Roots	-	-	-	-	-	-
others	-	-	-	-	-	-

Relationship between quantity of poison ingested and cardiotoxicity

The mean number of seeds / fruits ingested in no cardiotoxicity group was 2 and that in less severe Cardiotoxicity group was 4. The difference was statistically significant (p=0.001). The range, mean and standard deviation of quantity of poison in each of the groups is depicted in table 8.

Table No. 8 Quantity of poison ingested and cardiotoxicity

Cardiotoxicity	Range	Mean	Standard Deviation
No Cardiotoxicity`	1-5	2	1
Less severe Cardiotoxicity	1-10	4	4
Severe Cardiotoxicity	1-18	3	1

Relationship between method of ingestion of poison and cardiotoxicity

59.6% of those who had taken the poison in the crushed form had less severe cardiotoxicity compared to 31% in those who had taken the poison chewed. Cardiotoxicity were more observed in those who took in a crushed form and this difference was statistically significant at p = <0.05. The details are shown in the table below and Figure 10

Table No. 9 Method of ingestion and cardiotoxicity

	Cardiotoxicity					
	No cardiotoxicity		Less severe cardiotoxicity		Severe cardiotoxicity	
	Count	N %	Count	N %	Count	N %
crushed	11	21.2%	31	59.6%	10	19.2%

chewed	20	47.6%	13	31.0%	9	21.4%
swallowed	7	70.0%	2	20.0%	1	10.0%
others	-	-	-	-	-	-

Figure 9- Part ingested and Cardiotoxicity

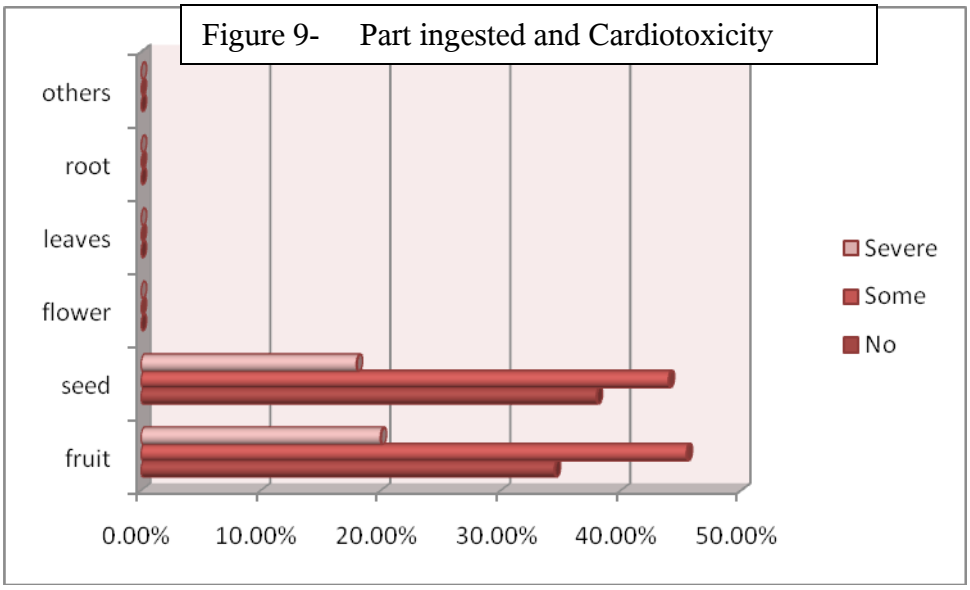
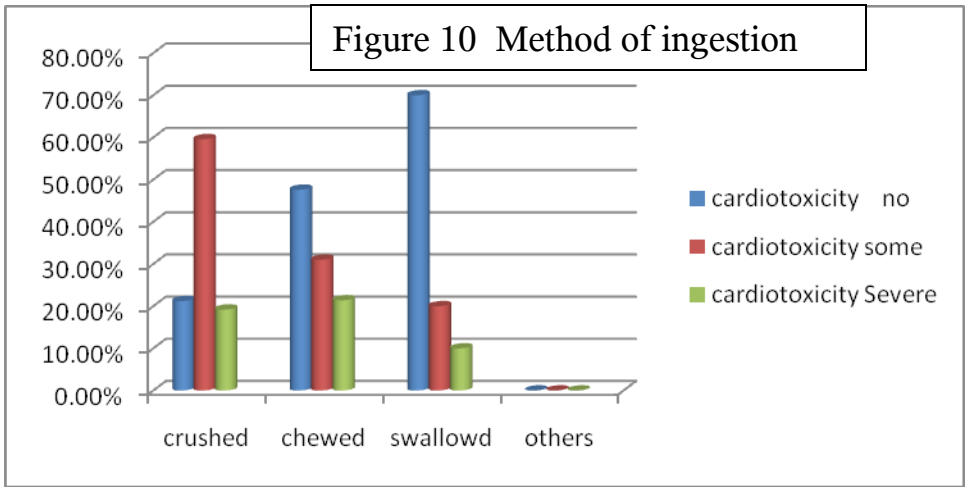


Figure 10 Method of ingestion



Relationship between manner of consumption and Cardiotoxicity

60.4% of the patients had consumed the poison in empty stomach and the rest after food. 47.7% of the patients who had taken the poison in empty stomach had some form of cardio toxicity compared to 16.2% in patients who took the poison after food. The difference between the groups was statistically significant ($p<0.05$). The details are shown in the graph and the table given below.

Table No. 10 Manner of consumption and cardiotoxicity

Manner of consumption	No Cardiotoxicity		Less severe cardiotoxicity		Severe cardiotoxicity	
	No	%	No	%	No	%
In empty Stomach	17	16.2%	35	33.3%	15	14.3%
After food	21	20.0%	12	11.4%	5	4.80%

Relationship between first aid and cardiotoxicity

Majority were not given first aid (57.7% of cases). 59.6% of patients who were given first aid developed some form of cardiotoxicity compared to 63.5% in patients who were not given first aid. (This refers to method adopted by local people to bring out the poison. The method mostly used was to induce vomiting using salt water, tamarind water, soap water etc). The difference between the groups was not statistically

significant (p=0.8529) The details are given in the table below and Figure 11

Table No. 11 First aid and cardiotoxicity

First aid	Cardiotoxicity					
	No Cardiotoxicity		Less severe cardiotoxicity		Severe cardiotoxicity	
	No	%	No	42.5	No	%
Given	13	12.4	21	20.0	9	8.6
Not Given	25	23.8	26	24.8	11	10.5

Relationship between time of first aid and cardiotoxicity

In patients who were given first aid 2 hrs after consumption of poison, all of them developed cardiotoxicity compared to 55% in patients who were given first aid within one hour. The difference between the groups was not statistically significant (p=>0.05) The details are given in the table below and explained using a graph(figure 12).

Table No.12 Time of first aid and cardiotoxicity

Time in minutes	cardiotoxicity					
	no		Less severe		severe	
	Count	percentage	Count	percentage	Count	percentage
1 to 60	9	45.0%	6	30.0%	5	25.0%
61 to 120	0	0%	2	66.7%	1	33.3%
>120	0	0%	5	83.3%	1	16.7%

Figure 11 First Aid

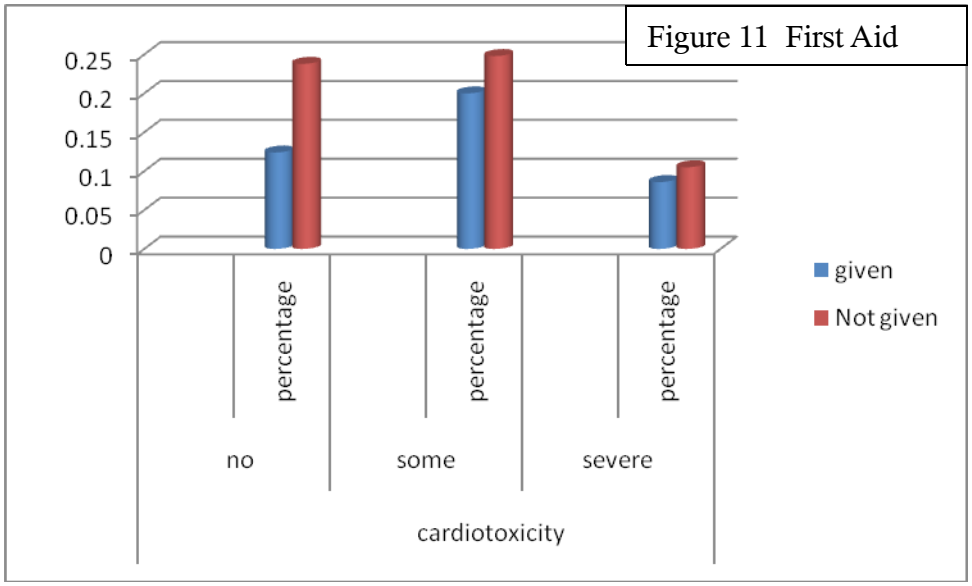
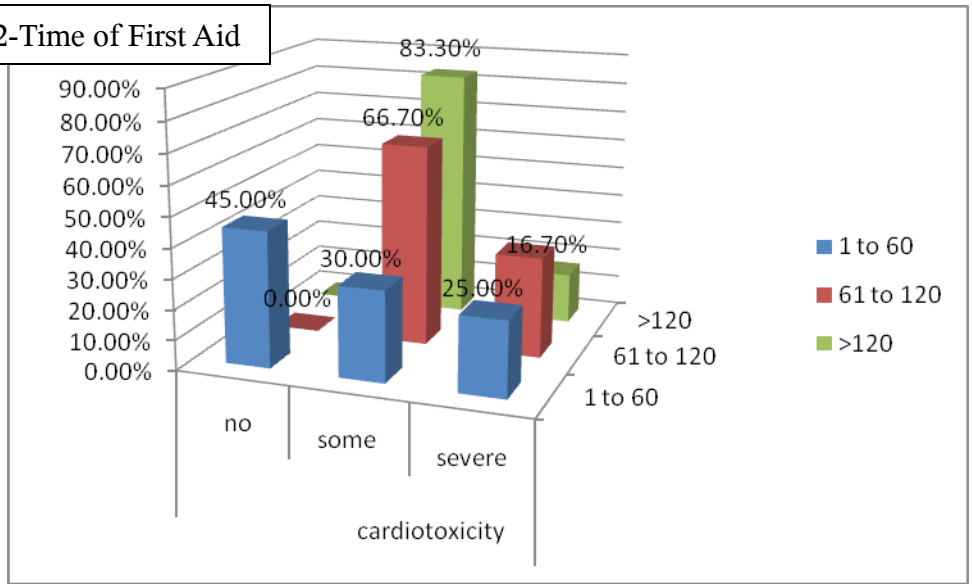


Figure 12-Time of First Aid



Relationship between consumption to admission interval and cardiotoxicity

The mean delay in getting to Coimbatore Medical College Hospital, Coimbatore after consumption of poison was 12.5±5.6hrs, the range being 1-62 hrs. The mean delay in no, less severe and severe cardiotoxicity group was 13.32, 12.25, and 12.36 hrs respectively. The difference between the groups was not statistically significant.

Relationship between symptoms and cardiotoxicity

The most common symptoms of yellow oleander poisoning were vomiting (72.6%), giddiness (56.6%) and diarrhea (35.8%).Approximately 75% of the patients who had at least two symptoms or all the three symptoms had features of cardiotoxicity. The difference between the groups was statistically significant (p=0.0005). The details are shown in the tables given below (table no. 13)

Table No.13 Symptoms of yellow oleander poisoning

Symptoms	No	%
Vomiting	77	72.6
Abdominal pain	16	15.1
Diarrhea	38	35.8
Giddiness	60	56.6
Numbness of tongue & lips	0	0
Altered mental status	0	0
Blurred vision	21	19.8
Palpitation	25	23.6
Shortness of sheath	8	7.5

Table No.14 Symptoms and cardiotoxicity

symptoms	cardiotoxicity		
	No	Less severe	severe

	Count	%	Count	%	Count	%
vomiting	22	21.0%	38	36.2%	16	15.2%
abdominal pain	6	5.7%	5	4.8%	4	3.8%
diarrhoea	7	6.7%	20	19.0%	10	9.5%
giddiness	17	16.2%	31	29.5%	11	10.5%
numbness of lips and tongue	0	0%	0	0%	0	0%
altered mental status	0	0%	0	0%	0	0%
blurring of vision	4	3.8%	10	9.5%	7	6.7%
palpitation	4	3.8%	11	10.5%	10	9.5%
shortness of breath	1	1.0%	4	3.8%	3	2.9%

Vital signs and biochemical parameters

The range, mean and standard deviation values of pulse rate, blood pressure and biochemical parameters are given in the table no.15. The mean values of all these parameters were within normal limits. Pulse rate was irregular at admission in 15.1% of the cases; in the rest the pulse rate was regular. Hypotension was present in two cases at admission.

Table No. 15 Vital signs and biochemical parameters

Parameter	Range	Mean	S.D
Pulse rate at admission	36-140	80	20
Systolic blood pressure (mm Hg)	70-150	112	14
Diastolic blood pressure (mmHg)	40-100	74	10
Blood urea (mg/dl)	15-72	27.5	10.44
Blood sugar (mg/dl)	56-235	93	31
Serum Creatinine (mg/dl)	0.6-1.8	.89	0.19
Na (meq/l)	122-160	138	5

Relationship between S.potassium and cardiotoxicity

The mean serum potassium values were higher in some (4.10 meq/l) and severe cardiotoxicity groups (4.10meq/l) compared to patients with no cardiotoxicity (3.9meq/I). There was a positive correlation between serum potassium levels and cardiotoxicity in the present study.

Table No.16 Serum K^+ levels in yellow oleander poisoning

	Cardiotoxicity					
	no		Less severe		severe	
S.potassium	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
	3.9	.4	4.1	.4	4.1	.4

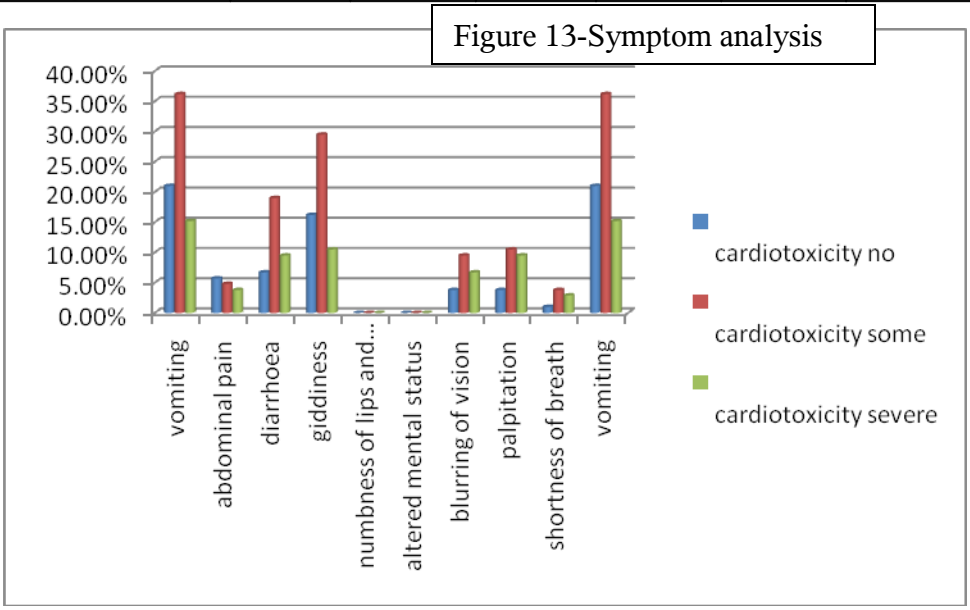
Relationship between gastric lavage and cardiotoxicity

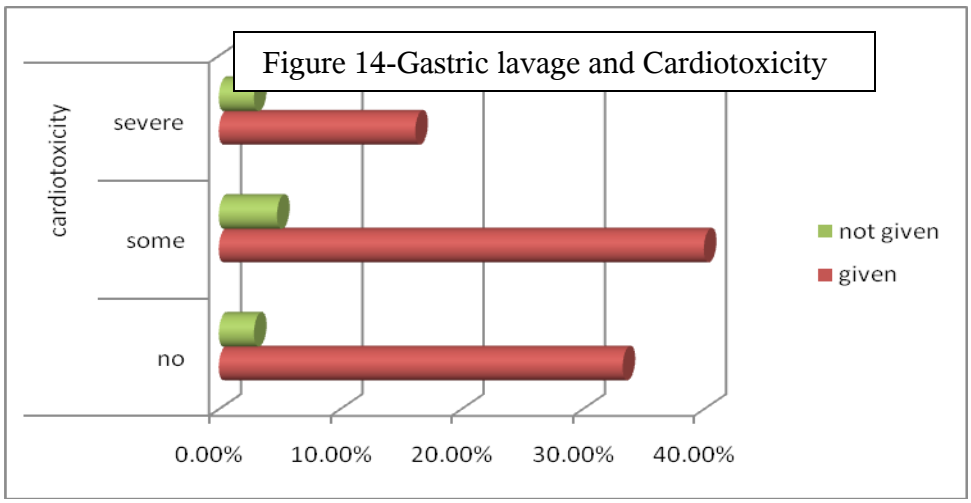
Gastric lavage was given in 89.2% of the cases. In those who were given gastric lavage, 33.3% did not develop any cardiotoxicity compared to 2.9% in those who were not given gastic lavage. The difference between the groups was statistically insignificant (p=>0.05). The table below shows the details.

Table No.17 Gastric lavage and cardiotoxicity

gastric lavage	cardiotoxicity					
	no		Less severe		severe	
	Count	N %	Count	N %	Count	N %
given	35	33.3%	42	40.0%	17	16.2%
not given	3	2.9%	5	4.8%	3	2.9%

Figure 13-Symptom analysis





Treatment given in yellow oleander poisoning

Most of the patients were given supportive treatment in the form of gastric lavage, injection atropine and tablet orciprenaline. Steroids were not used in any of the cases. Some patients were given tablet salbutamol and tablet deriphylline. Few patients received injection sodium bicarbonate. The details are given in the table given below.

Table No.18 Treatment given in yellow oleander poisoning

	No	%
Gastric lavage	95	89.6
Injection atropine	76	71.7
Tablet orciprenaline	93	87.7
Steroids	0	0

Relationship between cardiotoxicity and duration of hospital stay

The mean duration of hospital stay was 5 days, range being 1-9 days. The mean duration in no, less severe and severe cardiotoxicity groups was 4, 5 and 5days

respectively. The difference between the groups was statistically significant (p=0.0001). The details are given in the table below and explained using a graph.

Table No. 19 Duration of hospital stay and cardiotoxicity

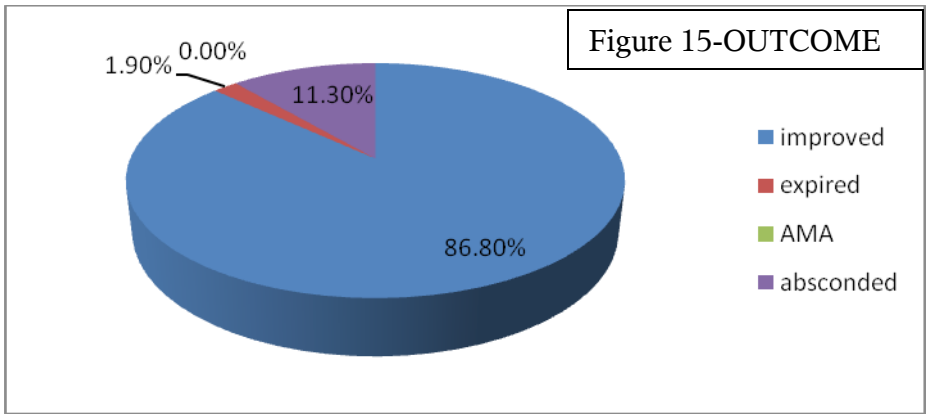
Duration of hospital stay	No Cardiotoxicity	Less severe cardiotoxicity	Severe cardiotoxicity
Mean	4	5	5
S.D.	1	1	2
Range	1-9		

Outcome

Out of 111 cases, 86.4% of the cases were discharged well. Death occurred in two cases (1.9%, one male and female patient) and 11.7% of the patients absconded from the wards. There was no statistically significant difference in outcome among males and females (p=0.7002)

Table No:20 Outcome

OUTCOME	count	percentage
improved	92	86.8%
expired	2	1.9%
AMA	0	.0%
absconded	12	11.3%



Relationship between cardiotoxicity and outcome

Out of 106 cases, only two deaths occurred. The deaths occurred in patients who had severe cardiotoxicity. But there was no statistically difference in outcome among no, some and severe cardiotoxicity groups (p=>0.05)

Table No 21 Cardiotoxicity and outcome

outcome	cardiotoxicity					
	no		Some		severe	
	Count	percentage	Count	percentage	Count	percentage
improved	29	44	18	29	44	18
expired	0	1	1	0	1	1
AMA	0	0	0	0	0	0
absconde d	9	2	1	9	2	1

DISCUSSION

Yellow oleander poisoning is a common method of deliberate self-harm among young adults in Srilanka and southern India. This study was done to find out the burden of this poisoning among admissions in general medicine wards of Coimbatore Medical College Hospital, Coimbatore, to analyse the clinical aspects, to correlate clinical and biochemical parameters with cardiotoxicity and to identify the possible risk factors for cardiotoxicity and outcome.

The incidence of yellow oleander poisoning among total number of admissions in general medicine wards of our hospital during the study period was 11.8 per 1000 admissions. Though it has been mentioned in previous studies that thousands of cases occur in Srilanka every year[4], the exact incidence in a particular population has not been previously reported. Among the total number of poisoning cases, yellow oleander accounted for 12.7% of the cases.

Among the 106 cases studied, the mean age of patients was 27+/ 10 years, range being 13-62 years (pediatric cases not included). 72.7% of the cases occurred in the age group between 13-29 years. This observation confirms the observation of previous Indian and Srilankan studies that yellow oleander poisoning was found commonly among adolescents and young adults[4,7]. Eddleston M et al, in a study of 415 cases in Srilanka observed that the patients were young (mean age 25.8 years, range 11-71). In that study more than 50% of women and 35% of men were under 21 years[4]. 49 (40.05%)

were males and 57 (54.95%) were females. The ratio of females: males was 1.16:1

Regarding the sex distribution 53.8% of the cases were females and 46.2% cases were males, the ratio being 1.16:1. In the present study there was only a slight female preponderance when compared to findings of previous Indian and Srilankan studies. Eddleston M et al, observed a female : male ratio of 1.6:1 in his study involving 415 patients in srilanka[4]. Generally this poisoning was found to be more common among females when compared to males[4,7,25].

Majority of the patients belonged to the upper lower and lower socioeconomic class (Kuppuswami, 1962 (modified)) 77.3% of the patients who were earning had income below Rupees two thousand per mth. The mean income was Rupees 1603 and majority were daily wage labourers.

75% of the patients were from the rural areas probably due to the fact that the plant can be easily found and widely grown in rural areas. There was no expenditure of money in consuming the poison and for many patients this was only poison known to them.

Most of the cases occurred during the working hours 6am – 6pm (84.9%) when the relatives were away for work or the victims have consumed the poison near their workplace.

The intention behind the poisoning was suicidal in 81.1% of the cases. The reasons included interpersonal conflict, unemployment, failure to achieve goal, situational reaction, grief reaction, physical illness etc. Two patient had underlying psychiatric illness and was on antipsychotic drugs. In 17% of the patients, poisoning attempt was done just to frighten others for some personal gain or to resolve conflicts. There were two cases of accidental poisoning among adolescents. There were no cases of homicidal poisoning probably due to the bitter taste of the poison. The observations regarding the intention behind poisoning were similar to that observed by Eddleston M et al[4].

Regarding underlying illness, except for three patients which includes two cases of psychiatric illness, one case of hypertension, none had any previous illness. Both of these patients were on irregular treatment. Three of the female patients were pregnant while consuming the poison and all of them recovered. This was similar to the findings observed by Eddleston M et al, who noted that only one patient had underlying illness (rheumatic heart disease)[8]. This observation differed markedly from patients with digoxin poisoning. Many of the patients with digoxin poisoning had underlying heart disease and were on multiple drugs.[27]. This observation is important because as a result of young age and previously healthy state, the cardiac arrhythmias induced by yellow oleander poisoning are unlikely to result from pre-existing conditions.

The electrocardiographic changes that were noted in this study was mainly due to depressed conduction. Most common abnormalities were sinus bradycardia, ST-T changes suggestive of digoxin effect toxicity, first-degree AV block, third-degree AV block and sino-atrial block. Others included second-degree AV block, junctional rhythm, AV dissociation and atrial ectopics. The observations were similar to that of Eddleston M et al except that tachyarrhythmias (0.5-1%) like atrial flutter, atrial fibrillation, ventricular tachycardia and ventricular fibrillation observed by Eddleston M et al were not observed in the present study. In the same study 3-6% had supraventricular tachycardia and 2% had ventricular ectopics which was not observed in the present study[8]. Mobitz type – II second-degree AV block which is not described in digoxin toxicity occurred in two cases[28]. In yellow oleander poisoning arrhythmias due to depressed conduction were more common than tachyarrhythmias and thus differs from digoxin poisoning in which tachyarrhythmias were found to be more common[27].

Depending on the ECG changes patients were divided into no, less severe and severe cardiotoxicity groups and the relationship between various factors and cardiotoxicity was studied (Refer definitions in materials and methods). Majority (63.8%) of cases showed some form of cardiotoxicity. Severe cardiotoxicity was present in approximately 19% of the cases.

The common method of poisoning in the present study was ingestion of fruits or seeds in the crushed or chewed form. Three patients had taken the outer fleshy

covering of the nut and these patients had no cardiotoxicity. Toxicological studies in albino rats have shown that all parts of the plant were poisonous especially the seeds / kernels of fruit[16]. Other parts like roots, leaves or flowers were not taken. The mean number of seeds / fruits ingested in severe (3) and less severe cardiotoxicity groups (4) were higher than that in no cardiotoxicity group (2) The range in no, some and severe cardiotoxicity groups were 1-5, 1-10, and 1-18 (seeds/fruits) respectively. Although there was a positive correlation between quantity of poison and cardiotoxicity in the present study it should be noted that even one seed / fruit was found to cause some or severe cardiotoxicity. Relationship between quantity of poison and outcome was not present as only two deaths had occurred and patients who had taken six and ten seeds had all survived. Two patients who died had consumed 5 and 18 seeds respectively. Eddleston M et al (1999) also observed no relationship with seeds ingested and outcome. In that study six patients who had died had consumed 10, 5,8,1,5 and 2 seeds[4]. So the quantity of poison alone cannot determine outcome, it may be influenced by other factors like method of ingestion, consumption in empty stomach or not, delay in gastric lavage, treatment given etc.

Majority had taken the seeds / fruits in the crushed form. 59.6% of those who had taken the poison in the crushed form had less severe cardiotoxicity compared to 31% in those who had taken the poison chewed, which shows a higher incidence of cardiotoxicity (statistically significant) in those who had taken the seeds / fruits crushed compared to those who had chewed or swallowed the poison. This is probably due to the

fact that more amount of cardiac glycoside is available to be absorbed once the seeds/fruits are crushed and one patient developed severe cardiotoxicity. This finding is supported by the fact that in srilanka people usually eat the seeds whole and they develop cardiotoxicity. So even the seeds taken as a whole can cause cardiotoxicity but to a lesser extent when compared to crushed or chewed form. The method of ingestion observed in the present study was similar to that reported in a study from Eastern India in which majority (97.33%) of the patients had ingested the poison in the crushed form[7].

Most of the patients (60.4%) had consumed the poison in empty stomach. There was a higher incidence of cardiotoxicity in patients who had consumed the poison in empty stomach than after food. This is probably due to the fact that absorption of cardiac glycoside is better in empty stomach. In previous studies this relationship was not assessed.

In 57.7% of cases first aid was given at home after the ingestion of poison. The commonly used method was to induce vomiting using soap water, tamarind water, salt water etc. There was no statistically significant difference in cardiotoxicity between patients who were given first aid and those who were not given first aid. This is because of the fact that in many patients there was a delay in giving first aid. But in patients who were given first aid, delay in giving first aid was associated with increased incidence of cardiotoxicity. So bringing out the poison early before the poison has passed into intestine reduces the incidence of cardiotoxicity in yellow oleander poisoning. The

relationship between time of first aid and cardiotoxicity was not studied in previous reports.

The mean delay in getting admitted to Coimbatore Medical College, Coimbatore after consumption of poison was 12.5±5.6 hrs, the range being 1-62 hrs. A correlation between delay in getting admitted to our hospital and cardiotoxicity could not be obtained probably due to the fact that many patients were given first aid and treated outside in other hospitals/institutions before being referred to our hospital. Bose TK et al (1999) observed a delay of 6-8 hrs[7]. The reasons for the delay in getting to our hospital in the present study would be treatment of patients outside by others doctors and lack of adequate transportation in interior rural areas. In patients with high suicidal intent the relatives come to know of the poisoning only after several hours and this night have contributed to the delay.

The most common symptoms of yellow oleander poisoning in the present study were vomiting (72.6%), giddiness (56.6%) and diarrhea (35.8%). This was similar to findings noted by Saravanapavanathan N and Ganeshmoorthy J (1986) who observed vomiting, giddiness and diarrhea as the most common symptoms in their study of 170 cases in Srilanka[2]. Patients who had vomiting, diarrhea or altered mental status had higher incidence of cardiotoxicity compared to those who did not have nay one of the above symptoms. Among patients who had at least two or all the three of above symptoms 75% developed cardiotoxicity. Thus patients with either vomiting, diarrhea or

altered mental status should be closely monitored for cardiotoxicity. Ellenhorn and Barceloux (1988) have also noted that in severe poisoning diarrhea and vomiting are early features[20].

The average pulse rate at admission was 80±20 per minute, range being 36-140 per minute. Many patients had a normal pulse rate and rhythm at admission only to develop features of cardiotoxicity later usually within a day.

Eddleston M et al (1999) have described a patient who remained in sinus rhythm for three days before developing second degree AV block[4]. So patients may have to be observed for 3-4 days after ingestion of poison before being discharged home.

Hyperkalemia occurs in severe yellow oleander poisoning[8]. Severe hyperkalemia can contribute to atrioventricular (AV) block and depressed myocardial excitability[19]. In the present study hyperkalemia was noted in only two our of 97 cases (2.1%) in whom the serum potassium levels were measured. Both of these patients had some cardiotoxicity. But there was a correlation between serum potassium levels and cardiotoxicity in the present study. The mean serum potassium values were higher in some (4.10 meq/l) and severe cardiotoxicity groups (4.10meq/l) compared to patients with no cardiotoxicity (3.9meq/I). Eddleston M et al (2000) noted hyperkalemia in 38 patients out of 118 cases (32.2%). Very high values of potassium like 7.2meq/l, 8.4 meq/l and 10.8 meq/l were observed in that study[8]. In the present study the two patients who had hyperkalemia had values of 5.5 and 5.2 meq/l. The reason why hyperkalemia

was not as common when compared to Srilankan study would be that poisoning might have been less severe or due to the persistent vomiting due to poisoning per se or due to induced emesis. Another reason would be that serial serum potassium measurements were not obtained due to technical constraints and hyperkalemia developing during the course of poisoning was missed (serum potassium levels were measured only at admission). Moreover the baseline potassium level in the population also was not known. Hypokalemia was noted in 6 of our cases. Eddleston M et al, (2000) noted hypokalemia in 9 out of 118 cases[8]. Hypokalemia may be probably due to persistent vomiting due to poisoning per se or due to induced emesis. Hypokalemia can exacerbate cardiac glycoside toxicity as if facilitates enhanced binding of cardiac glycosides to Na^+ -K^+ ATPase pump[22]. Both hyperkalemia and hypokalemia are dangerous in yellow oleander poisoning and serial monitoring of potassium levels and adequate treatment is necessary. Severe hyperkalemia may require treatment with antidigoxin Fab fragments[18].

There was no statistically significant difference in cardiotoxicity between patients who were given gastric lavage and those who were not given gastric lavage. Gastric lavage is most useful when started within 60 minutes after ingestion[24]. Early gastric lavage is more important than whether gastric lavage is given or not in reducing the incidence of cardiotoxicity. This relationship was not assessed in previous published reports.

Most of the patients were treated with supportive measures like gastric lavage, injection atropine, tablet orciprenaline and none of the cases steroids were used. There are no scientific studies or trials to support the use of steroids in yellow oleander poisoning. Activated charcoal, temporary cardiac pacing and antidigoxin Fab fragments were not used due to economical and technical constraints.

The mean duration of hospital stay in the present study was 5 days, range being 1-9 days. Those patients with some and severe cardiotoxicity had increased duration of hospital stay when compared to patients with no cardiotoxicity. This was similar to findings observed by Boss TK et al (1999) who observed a median hospital stay of 5 days[7].

Death occurred in two cases (one male and female patient) within one hour after admission. The male patient had atrioventricular dissociation while the female patient died before ECG could be taken. The case fatality rate was 1.88%. Bose TK et al (1999) observed a case fatality rate of 4.6% among 300 patients in eastern India[7]. In Srilankan studies, Eddleston M et al observed a case fatality rate of approximately 10%[4]. The lower case fatality rate in the present study may be due to les severe poisoning in Tamilnadu when compared to that in Srilanka and probably due to lesser number of patients studied. As the death were very few, there was no statistically significant difference in outcome among no, some and severe cardiotoxicity groups and also there was no statistically significant difference in outcome among male and female patients.

Areas of further work

1. The efficacy of anti – digoxin Fab fragments in treating serious cardiac arrhythmias induced by yellow oleander has been established by Eddleston M et al (2000) in the only small randomized clinical trial involving 66 patients[10]. Further studies are required to confirm the efficacy of anti-digoxin Fab fragments in treating serious cardiac arrhythmias induced by yellow oleander.

2. Rapid detection of oleander poisoning with Digoxin III, a new digoxin assay as shown by Dasgupta A, Risin SA, Reyes M, Actor JK (2008)[33].Further studies with regard to this is necessary to confirm its use in rapid detection of yellow oleander poisononing.

3. Multiple doses of activated charcoal was found to be safe and more effective than single does activated charcoal in reducing death and life threatening cardiac arrhythmias after yellow oleander poisoning in the only single-blind, randomized placebo-controlled trial in Srilanka[9,31,36]. Further studies are required to confirm the superiority of multiple doses of activated charcoal over single dose activated charcoal in yellow oleander poisoning . Further studies are required to confirm the superiority of multiple doses of activated charcoal over single dose activated charcoal in yellow oleander poisoning.

4. Focal myocardial edema has been noted in cases of yellow oleander poisoning[7]. So studies can be taken up to assess whether glucocorticoids have any role in improving outcome in this poisoning.

CONCLUSIONS

1. The incidence of yellow oleander poisoning in general medicine wards of Coimbatore Medical College Hospital ,Coimbatore during the study period was 11.8 per 1000 admissions.

2. Yellow oleander poisoning was most commonly observed among young adults and adolescents.

3. Although there was a slight female preponderance the difference was not statistically significant.

4. Most of the patients were from rural areas belonging to upper lower and lower socio-economic class.

5. Most of the cases occurred during day time (6am – 6pm).

6. In majority of cases, the intention was suicidal secondary to interpersonal conflict, grief reaction, situational reaction, unemployment etc.

7. The most common symptoms of yellow oleander poisoning in the present study were vomiting, giddiness and diarrhea.

8. Electrocardiographic changes noted in the present study were mainly due to depressed conduction. Most common abnormalities were sinus bradycardia, ST – T changes similar to digoxin effect / toxicity, first – degree AV block, third –

degree AV block and sino-atrial block.

9. Hyperkalemia as a manifestation of yellow oleander poisoning was uncommon in the present study compared to Srilankan studies.

10. There was a higher incidence of cardiotoxicity as the quantity of poison increased but even ingestion of one seed / fruit was found to cause severe cardiotoxicity.

11. There was a higher incidence of cardiotoxicity in those who had taken the seeds / fruits crushed when compared to those who had chewed or swallowed the poison.

12. Cardiotoxicity was found to be higher in patients who had consumed the poison in empty stomach than after food.

13. Delay in inducing emesis or giving gastric lavage was associated with increased incidence of cardiotoxicity.

14. The occurrence of cardiotoxicity was higher in patients who had vomiting, diarrhea or altered mental status compared to patients who did not have any of these symptoms.

15. The mean serum potassium values at presentation were higher in patients who had cardiotoxicity when compared to patients who had no cardiotoxicity.

16. Case fatality rate was low in the present study when compared to previous Srilankan and Indian studies.

SUMMARY

Yellow oleander poisoning is a common method of suicide in South India and Srilanka with thousands of cases occurring in Srilanka each year at present. This study was undertaken to find out the incidence of yellow oleander poisoning in our hospital, to study the clinical aspects, to find out correlation between clinical and biochemical parameters with electrocardiographic changes and to find out possible risk factors for cardiotoxicity and outcome.

After institutional ethical clearance, with an informed consent and with inclusion and exclusion criteria 106 cases of yellow oleander poisoning were included in the study and were evaluated on clinical, biochemical and electrocardiographic aspects. The data were entered in Microsoft Excel spread sheet and analysed statistically in SPSS software

The incidence of yellow oleander poisoning in general medicine wards of our hospital was 11.8 per 1000 admissions. 53.8% of the cases were females. The mean age of poisoning was 27$\pm$ 10 years. The intention was suicidal in majority. The most common symptoms were vomiting, numbness of tongue and lips, giddiness and diarrhea. The most common abnormal ECG findings were sinus bradycardia, ST – T changes similar to that described for digoxin effect / toxicity, sino-atrial block, first – degree AV block and third – degree AV block. Depending on the ECG changes, patients were decided into no, some and severe cardiotoxicity groups and relationship between various factors and cardiotoxicity analysed. The mean number of seeds / fruits ingested in

patients who had cardiotoxicity was higher than in patients who had no cardiotoxicity. Majority had taken the poison crushed and in empty stomach which resulted in greater cardiotoxicity. Delay in inducing emesis or giving gastric lavage was associated with greater cardiotoxicity. The occurrence of cardiotoxicity was higher in patients who had vomiting, diarrhea or altered mental status. The mean serum potassium values at presentation were higher in patients who had cardiotoxicity. Hyperkalemia was noted in only two cases. The mean duration of hospital stay was 5 ± 1.5 days and was higher in patients who had cardiotoxicity. The case fatality rate was 1.88%.

Yellow oleander poisoning was found to be a common method of suicide among young adults and adolescents in this part of the country and was slightly more common in females. It accounted for 13.1% of cases among the total number of poisoning cases during the study period. Most of the patients were from rural areas belonging to upper lower and lower socio – economic class. The most common symptoms were vomiting, numbness of tongue and lips, giddiness sand diarrhea. The most common electrocardiographic abnormalities were sinus bradycardia, ST – T changes similar to digoxin effect ./ toxicity, first-degree AV block, third-degree AV block and sino-atrial block. Hyperkalemia was found to be uncommon when compared to previous Srilankan studies. The occurrence of cardiotoxicity was greater in patients who had taken the poison crushed and in empty stomach, who had taken more quantity of poison, in whom there was a delay in inducing emesis or giving gastric lavage, who had vomiting, diarrhea or altered mental status and who had higher serum potassium levels at

admission. The more severe the cardiotoxicity more was the duration of hospital stay. The case fatality rate was low when compared to previous studies.

Although this study could identify the risk factors for cardiotoxicity, the risk factors associated with poor outcome could not be well established as the deaths were very few. Hence further studies are required to identify risk factors associated with poor outcome in this poisoning. Further studies are also required to assess the role of glucocorticoids in improving outcome. The benefit reported with multiple doses of activated charcoal and anti-digoxin Fab fragments also need to be confirmed by further studies.

BIBLIOGRAPHY

1. Driggers DA, Solbrig R, Steiner JF, et al : Acute oleander poisoning – A suicide attempt in a geriatric patient. West J Med 1989 Dec; 151 : 660 – 662.

2. Saravanapavananthan N and Ganeshamoorthy J (1988) Yellow oleander poisoning – a study of 170 cases. Forensic Science International 36, 247 – 250.

3. Sreeharan N, Putharasingam S, Ranjadayalan K, et al. Yellow oleander poisoning – Clinical manifestations and prognostic criteria. Jaffna Medical Journal 1985 ; 100, 101.

4. Eddleston M, Ariaratnam CA, Meyer PW, et al. Epidemic of self poisoning with seeds of the yellow oleander tree (Thevetia peruviana) in northern Srilanka. Trop Med Int Health 1999 ; 4:266-73.

5. Modi NS Modi's Textbook of Medical Jurisprudence and Toxicology. 22nd Edition (Editor Subrahmanyan BV). Butterworths India, New Delohi, 1999.

6. Pearn J, Oleander poisoning. In : Toxic Plants and Animals : A guide for Australia. 2nd Edition (Eds Covacevich J, Davine P and Pearn J). William Brooks, Brisbane, 1989 : 37-50.

7. Bose TK, Basu RK, Biswas B, et al. Cardiovascular effects of yellow oleander ingestion. J Indian Med Assoc. 1999 Oct; 97 (10) : 407 – 410.

8. Eddleston M, Ariaratnam CA, Sjostrom L, et al. Acute yellow oleander (Thevetia peruviana) poisoning : Cardiac arrhythmias, electrolyte disturbances and serum cardiac glycoside concentrations on presentation to hospital. Heart 2000 ; 83:301-306.

9. de Silva HA, Fonseka MM, Pathmeswaran A, et al. Multiple – dose activated charcoal for treatment of yellow oleander poisoning : A single – blind, randomized, placebo – controlled trial. Lancet 2000, Jun 7 ; 363 (9373) : 1935 – 38.

10. Eddleston M, Rajapakse S, Rajakanthan, et al. Anti – digoxin Fab fragments in cardiotoxicity induced by ingestion of yellow oleander : a randomized control trial.

11. Aggarwal A. The poison sleuths death by yellow kaner. Science Reporter, 1999 Sep; 9 : 31-34.

12. Middleton WS, Chen KK. Clinical results from oral administration of thevetin, a cardiac glycoside. Am Heart J 1936 ; 11:75-88.

13. Radford DJ, Gillies AD, Hinds JA, Duffy P. Naturally occurring cardiac glycosides. Med J Aust 1996 ; 144 : 540 – 544.

14. Watt MW, Breyer p Brandwijk MG. The medicinal and poisonous plants of southern and eastern Africa. Edinburgh, E&S Livingston, 1962 : 107-9.

15. Langford SD, Boor PJ. Oleander toxicity : an examination of the human and animal toxic exposures. Toxicology 1996 ; 109 :1-13.

16. Oji O, Okafor QE. Toxicological studies on stembark, leaf and seed kernel of yellow oleander (Thevetia peruviana). Phytother Res 2000 Mar ; 14(2) : 133-5.

17. Arnold HL, Middleton WS, Chen KK. The action of Thevetin, a cardiac glycoside and its clinical application. American J Med Sci. 1935 : 189 – 193.

18. Douglas RL, Horowitz BZ, Hodgman M, et al. Plant poisoning, Glycosides – cardiac. www.emedicine.com/emerg/topic 439.htm.

19. Haddard ML, Shannon WM, Winchester FJ. Clinical Management of Poisoning and Overdose. 3rd edition, W.B. Sanders Company, 1998.

20. Ellenhorn MJ & Braceloux DG (1988). 1st edn. Medical Toxicology – Diagnosis and treatment of human poisoning, New York, Elsevier Science Publishing

Company Inc. 1252 – 1255.

21. Duke JA (1987) edn. Handbook of Medicinal Herbs, USA, CRC Press, Inc.

22. Tripathi KD, Essentials of Medical Pharmacology, 5th Edition. Jaypee Brothers, New Delhi, 2003.

23. Osterlok J, Herold S, Pond S : Oleander interference in the digoxin radioimmunoassay in a fatal ingestion. JAMA 1982 ; 247:1596-1597.

24. Tierney Jr. CM, McPhee JS, Papadakis MA (Editors), Current Medical Diagnosis and Treatment 2005. 44th edn. Lange Medical Books / McGraw – Hill.

25. Flanagan RJ, Jones AL. Fab antibody fragments : Some applications in clinical toxicology. Drug Saf. 2004 ; 27(14) : 1115 – 33.

26. Fonseka MM, Seneviratne SL, de Silva CE, et al. Yellow oleander poisoning in Srilanka : Outcome in a secondary care hospital. Hum Exp Toxicol 2002 Jun ; 21(6) : 293 – 95.

27. Gaultier M, Fournier E, Efthymiou ML. Intoxication digitalique aigue (70 observations). Bull Soc Med Hop Paris 1968 ; 119 : 247.

28. Schamroth L. An Introduction to Electrocardiography (Editor Schamroth C). Seventh Edn, 1990, Blackwell Science Ltd.

29. Efficacy of activated charcoal in yellow oleander poisoning. Ceylon Med J. 2008 Jun;53(2):33-5.

30. A fatal case of oleandrin poisoning. Forensic Sci Int. 2008 Aug 6;179(2-3):e31-6. Epub 2008 Jul 7..

31. Multiple-dose activated charcoal in yellow oleander poisoning. Lancet. 2008 Jun 28;371(9631):2171

32. Rapid detection of oleander poisoning by Digoxin III, a new Digoxinassay: impact on serum Digoxin measurement. Am J Clin Pathol. 2008 Apr;129(4):548-53

33. Pharmacokinetics of digoxin cross-reacting substances in patients with acute yellow Oleander (Thevetia peruviana) poisoning, including the effect of activated charcoal.The Drug Monit. 2006 Dec;28(6):784-92.

34. Patterns of hospital transfer for self-poisoned patients in rural Sri Lanka: implications for estimating the incidence of self-poisoning in the developing world. Bull World Health Organ. 2006 Apr;84(4):276-82. Epub 2006 Apr 13

35. Nerium oleander self poisoning treated with digoxin-specific antibodies]. Ann Fr Anesth Reanim. 2005 Jun;24(6):640-2

36. Multiple-dose activated charcoal for treatment of yellow oleander poisoning : a single-blind, randomised, placebo-controlled trial. Lancet. 2003 Jun 7;361(9373):1935-8.

37. Nerium oleander poisoning .Rev Clin Esp. 2007 Nov;207(10):53

DEPARTMENT OF GENERAL MEDICINE , COIMBATORE MEDICAL COLLEGE HOSPITAL.

A STUDY OF CLINICAL ,BIOCHEMICAL AND ELECTROCARDIOGRAPHIC ASPECTS OF YELLOW OLEANDER POISONING

Informed consent form for prospective participants

Principal Investigator: Dr Abhishek S, Junior Resident.

Research Guide: Prof. Dr. Ramasamy.MD. Chief, Medical Unit – IV.

Organization: Department of Medicine, Coimbatore Medical College Hospital.

This informed consent form has two parts

PART – I INFORMATION SHEET(to share the information about the research with you)

PART – II CERTIFICATE OF CONSENT (for signatures if you agree to take part)

(You will be given a copy of the full informed consent form.)

PART – I INFORMATION SHEET

I , Dr Abhishek S, Junior resident in Dept of Medicine invites you to join as participant in my research on Yellow oleander poisoning, which is a common poisoning in our state. I am going to give you information and invite you to be part of this research. You do not have to decide today whether or not you will participate in the research. Before you decide, you can talk to anyone you feel comfortable with about the research.

There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take time to explain. If you have questions later, you can ask them to me, the study doctor or the staff.

Yellow oleander poisoning results in a variety of manifestations and as it is a cardiac toxin ,majority of it impacts are on the cardiovascular system in the form of conduction defects .It can also result in variations of ions like potassium and sodium which is normally present in the body. We are doing this research to learn the various aspects this poisoning in our population and to identify various risk factors so that we can have better understanding of this poisoning and to compare with other population type and finally treatment efforts in a better way.

In this study you will have to answer questions regarding your illness, undergo a physical examination , give blood for tests, undergo an electrocardiogram.

You are being selected because we are inviting all adults with acute yellow oleander poisoning to enroll in the study.

Your participation in this research is entirely voluntary. It is your choice whether to participate or not. Whether you choose to participate or not, all the services you receive at this clinic will continue and nothing will change. You may change your mind later and stop participating even if you agreed earlier.

You will have to give details regarding your age,sex,occupation,time/place/quantity of poison consumption,regarding treatment obtained outside if so, duration of disease, family ,past medical/ mental illness symptoms you are having at present, and current medications. A doctor will examine you to look for any problems. Your general condition will be recorded. All the data will be recorded in a proforma. Ten ml of blood will be drawn for doing various laboratory tests to know about the status of your disease. Any excess sample will be destroyed immediately after the laboratory tests are completed. Taking the blood sample will produce some pain and there may be slight redness at the site of puncture for a day or two. You will be subjected to a electrocardiogram(ECG) which will be recorded and it is a painless procedure.

On the first day you will be asked about your problems, a doctor will check you up and an ECG will be taken. You will also have to give the blood sample for examination.

If you participate in this research you will be having a thorough check up, which may reveal some unidentified problems in you. We will promptly start the treatment for them. Also by participating you are providing valuable data that will help doctors understand this disease better and ultimately serve the patients in a better way.

We will not be providing any money for participating in this research, you may incur more expense since you will have to visit the hospital more frequently.

It is possible that if others in the community are aware that you are participating in this research, they may ask you questions. We will not be sharing the identity of those participating in the research with anyone. The information that we collect from this research project will be kept confidential. Information about you that will be collected during the research will not be identified by your name but by a number. Only the researchers will know what your number is and they will lock that information up with a lock and key. It will not be shared with or given to anyone except my research guide.

The knowledge that we get from doing this research will be shared with you before it is made widely available to the public. Confidential information will not be shared. There will be small meetings in the community and these will be announced. After these meetings, we will publish the results in order that other interested people may learn from our research

You do not have to take part in this research if you do not wish to do so. You may also stop participating in the research at any time you choose. It is your choice and all of your rights will still be respected

If you have any questions you may ask them now or later, even after the study has started. If you wish to ask questions later, you may contact

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This proposal has been reviewed and approved by the Ethics Committee of Coimbatore Medical College Hospital which is a committee whose task it is to make sure that research participants are protected from harm.

PART – II CERTIFICATE OF CONSENT

I have been invited to participate in research on diabetes. I understand that it will involve answering a detailed questionnaire, undergoing a thorough physical exam, giving blood and urine samples and two or three follow-up visits. I have been informed that the risks are minimal and may include only slight pain and redness at sight of needle prick. I am aware that there may be no benefit to me personally and that I will not be compensated monetarily. I have been provided with the name of a researcher who can be easily contacted using the number and address I was given for that person.

I have read the foregoing information or it has been read to me. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research and understand that I have the right to withdraw from the research at anytime without in anyway affecting my medical care.

Name of the participant: _____

Signature of the participant: _____

Date: _____
(Day/Month/Year)

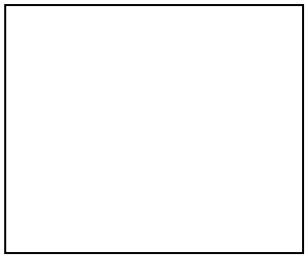
<u>If illiterate</u>

A literate witness must sign (if possible , this person should be selected by the participant and must have no connection to the research team)
I have witnessed the accurate reading of the consent form to the potential participant , translated to his mother tongue, and the individual has had opportunity to ask questions. I confirm that the individual has given consent freely.

Name of witness: _____ AND Thumb print of participant

Signature of witness: _____

Date : _____
(Day/Month/Year)



I have accurately read or witnessed the accurate reading of the consent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of the researcher: _____

Signature of the researcher: _____

Date: _____
(Day/Month/Year)

PROFORMA

CLINICAL,BIOCHEMICAL AND ELECTROCARDIOGRAPHIC ASPECTS OF YELLOW OLEANDER POISONING

Name Age Sex IP No

Occupation Income

DOA DOD Duration of Hospital stay

Religion

Marital Status

Address

SOCIAL PROFILE

Place of ingestion :

Part ingested : leaves/flower/fruit/seeds/roots/others

Quantity of Poison :

Method of ingestion:

Mixed with :

Consumption : Empty stomach/ After food( )hrs

Intention :suicide/Accidental/Homicidal/others

Reason for ingestion:

TIME PROFILE

Time of ingestion:

Time after which family members came to know

First aid at home Time

Treatment by other doctors/hospital Time

Time of admission

Underlying Psychiatric Disease Y/N if yes.

Other diseases:

Women – LMP Pregnancy Y/N

CLINICAL PROFILE

GI - Nausea/Vomiting/Abdominal pain/Diarrhoea/Anorexia

CNS- Giddiness/Headache/fatigue/weakness/numbness of tongue and lips/altered mental status/ seizures

Visual- Blurred vision / scotomas / flashes of light /Xanthopsia

Cardiac- Palpitations/shortness of birth/chest pain/chest discomfort

PHYSICAL EXAMINATION

Pulse rate Rhythm Volume

BP RR febrile/ afebrile Dehydration – No/some/severe

Pallor/icterus/cyanosis/clubbing/LNE/pedal oedema

CNS- level of consciousness

Tone DTR Plantar

INVESTIGATIONS

E

CG ALL LEADS

BLOOD UREA

RANDOM BLOOD SUGAR

S CREATININE

S SODIUM

S POTASSIUM

RHYTHM

STRIP

TREATMENT GIVEN

Gastric Lavage -Y/N

Activated Charcoal - Y/N

Inj Atropine - Y/N

T.Orceprenaline - Y/N

Steroids -Y/N

Others -Y/N Specify

OUTCOME -- IMPROVED / EXPIRED /Against Medical Advice /
ABSCONDED/Discharge at request

MASTER CHART KEY

YELLOW OLEANDER POISONING

1.Sex Male- 1 Female – 2

2. Marital status Married- 1 Unmarried – 2

Divorcee- 4 Widow – 5 Widower- 6

3. Part ingested Fruit- 1 Seed/Kernel -2 Flower -3

Leaves – 4 Root- 5 Other parts- 6

4. Method of ingestion Crushed - 1 Chewed – 2

Swallowed – 3 Others – 4

5. Consumption in Empty stomach – 1 After food – 2

6. Intention Suicidal -1 Accidental-2

Homicidal – 3 Others -4

7. First Aid Given – 1 Not given - 2

8. Treatment by other Doctors Yes – 1 No- 2

9. Vomiting Yes – 1 No- 2

10. Abdominal pain Yes – 1 No- 2

11. Diarrhoea Yes – 1 No- 2

12. Giddiness Yes – 1 No- 2

13. Numbness of tongue and lips Yes – 1 No- 2

14. Altered Mental status Yes – 1 No- 2

15. Blurred Vision Yes – 1 No- 2

16. Palpitations Yes – 1 No- 2

17. Shortness of Breath Yes – 1 No- 2

18. Pulse rhythm Regular- 1 Irregular - 2

19. Sinus rhythm Yes – 1 No- 2

20. Sinus Bradycardia Yes – 1 No- 2

21. Sinus Arrest or exit block Yes – 1 No- 2

22. Atrial ectopics Yes – 1 No- 2

23.Junctional Rhythm Yes – 1 No- 2

24.AV dissociation Yes – 1 No- 2

25. I deg AV block Yes – 1 No- 2

26. II deg AV block Mobitz type I -1 Mobitz type 2 - 3

27. III deg AV block Yes – 1 No- 2

28.ST- T changes Yes – 1 No- 2

29.Gastric Lavage Yes – 1 No- 2

30. Inj Atropine Yes – 1 No- 2

31. Tab Orciprenaline Yes – 1 No- 2

32. Steroids Yes – 1 No- 2

33. Outcome Improved – 1 Expired – 2

Against medical advice – 3 Absconded – 4

Sl no	name	age class	age	sex	marital st	incomed	income	hosp stay	part ing	quantity	methings	consump	intentn	time	timeingst	timefrsai	timeintrv	firstaid	trtm outs	vomiting	abdmnlp	diarrhoea	giddiness	numbnes	mentalst	vision	palptatn
1	rajesh	1	16	1	2	9	9	1	2	1	2	1	4	1	8	30	1	1	4	1	2	2	2	2	2	2	1
2	suja	1	14	2	2	9	9	6	2	1	1	2	1	2	20	.	.	2	15	2	1	1	1	2	2	2	2
3	raghu	1	20	1	2	2	1800	7	1	5	2	2	4	2	21	360	3	1	61	1	2	1	1	2	2	2	2
4	rajila	1	18	2	1	9	9	7	1	3	1	1	1	1	10.3	30	1	1	3	1	2	1	1	2	2	1	1
5	sajila	1	17	2	2	9	9	6	1	5	1	2	1	1	11.3	60	1	1	3	1	2	1	1	2	2	2	2
6	ummathal	3	30	2	1	1	800	2	1	1	2	2	1	1	10	30	1	1	2	1	2	2	2	2	2	2	2
7	sangar	3	31	1	3	1	1000	6	2	3	2	2	1	1	17.3	.	.	2	19	1	2	1	1	2	2	2	2
8	raju	2	25	1	2	3	2400	4	2	6	1	1	1	1	11	.	.	2	12	1	2	2	2	2	2	2	2
9	suresh	2	28	1	1	4	3000	5	2	4	2	1	1	1	14	.	.	1	1	1	2	1	1	2	2	2	2
10	chinnan	1	20	1	1	2	1125	4	2	4	2	1	1	1	8.3	.	.	2	.	1	2	1	1	2	2	1	2
11	suresh	1	18	1	2	3	2000	5	1	25	1	1	4	1	10.3	.	.	2	4	1	2	2	2	2	2	2	2
12	reema	2	27	2	1	9	9	6	2	3	1	1	4	1	11	60	1	1	3	1	2	1	1	2	2	2	2
13	sajeena	2	27	2	1	1	900	5	1	3	1	1	1	1	13	120	2	1	4	1	2	1	1	2	2	2	2
14	kumar	2	23	1	2	2	1800	3	2	2	3	2	1	1	11	.	.	2	2	2	2	2	2	2	2	2	2
15	meena	2	24	2	5	1	500	4	2	4	2	1	1	1	7	30	1	1	5	1	2	1	1	2	2	2	2
16	princy	2	22	2	1	9	9	4	2	3	1	2	1	1	18	.	.	2	4	1	2	2	2	2	2	2	2
17	saju	2	26	1	1	4	3000	6	2	5	2	2	1	1	12	15	1	1	2	2	2	1	1	2	2	1	2
18	nataraj	4	45	1	1	2	1500	5	1	6	9	1	1	1	13	10	1	1	3	1	2	1	1	2	2	2	2
19	mailal	2	21	2	1	1	300	4	1	1	1	1	4	1	8.15	.	.	2	2	1	2	2	2	2	2	2	2
20	suresh	4	40	1	1	1	600	6	2	4	2	1	1	2	5.2	130	3	1	10	1	2	2	2	2	2	2	2
21	rajesh	1	16	1	2	9	9	4	1	2	3	2	4	1	17	60	1	1	6	1	2	2	2	2	2	2	2
22	siva	1	15	1	4	1	500	6	1	6	1	1	1	2	5	.	.	2	16	1	2	1	1	2	2	2	2
23	agil	2	28	1	2	1	500	3	1	2	2	1	1	1	18	.	.	2	4	1	1	1	1	2	2	1	2
24	geeta	2	25	2	3	9	9	5	1	4	1	1	1	1	12	360	3	1	7	1	2	1	1	2	2	2	2
25	veena	3	32	2	1	1	600	4	2	3	3	1	4	2	23	.	.	2	11	1	2	1	1	2	2	1	2
26	rani	2	27	2	1	5	6000	5	2	5	2	1	1	1	15	.	.	2	5	1	2	1	1	2	2	2	2
27	sheela	2	28	2	2	2	1500	3	2	2	2	2	1	2	21	15	1	1	.	2	2	2	2	2	2	2	2
28	nataraj	4	45	1	1	3	2500	6	1	3	1	2	4	1	14	.	.	2	2	2	2	2	2	2	2	2	2
29	sivan	2	25	1	1	2	1800	2	1	1	1	1	1	1	8	.	.	1	.	2	2	2	2	2	2	2	2
30	kamal	1	19	1	2	2	1800	2	2	3	3	2	4	2	20.3	10	1	2	.	2	2	2	2	2	2	2	2
31	seeta	2	25	2	1	9	9	3	1	1	1	2	1	1	11.3	.	.	2	4	1	2	2	2	2	2	2	2
32	manju	2	28	2	1	1	600	4	1	1	2	1	1	1	11	.	.	1	.	1	2	2	1	2	2	2	2
33	anuja	1	20	2	2	9	9	3	1	3	1	2	1	1	10.3	15	1	2	8	1	1	2	1	2	2	2	1
34	aarthi	3	32	2	1	9	9	6	1	3	2	1	1	1	16	.	.	2	5	1	2	1	2	2	2	2	2
35	jancy	4	42	2	1	2	1800	1	1	1	2	2	4	1	14	.	.	1	2	2	2	2	2	2	2	2	2
36	saju	3	39	1	1	3	2100	2	2	2	2	1	1	1	8.3	30	1	2	7	1	2	2	2	2	2	2	2
37	liju	1	19	1	2	2	1500	2	1	1	3	2	4	1	9	15	1	2	4	2	2	2	2	2	2	2	2
38	kritka	1	20	2	1	9	9	5	2	3	1	2	1	1	6	30	1	1	4	1	1	2	2	2	2	2	2
39	deepak	3	30	1	1	3	2000	5	2	5	1	1	1	1	17	.	.	1	.	1	2	1	1	2	2	1	2
40	tamil	1	17	2	2	2	1650	6	1	3	1	1	1	1	17	60	1	1	5	1	2	1	1	2	2	2	1
41	sai	3	32	2	1	9	9	4	1	2	2	1	1	1	14	.	.	2	1	1	1	2	1	2	2	1	2
42	thangam	2	26	1	2	2	1500	6	2	3	1	2	1	2	4	15	1	1	17	1	2	1	2	2	2	2	2
43	preena	2	21	2	3	9	9	4	1	3	1	1	1	1	10	420	3	2	14	1	2	2	1	2	2	2	2
44	manoj	2	25	1	2	3	2000	5	2	5	1	2	1	1	13.3	.	.	1	23	1	2	2	2	2	2	2	2
45	malliga	2	25	2	1	1	200	4	1	2	2	1	1	1	17	.	.	1	5	2	2	2	1	2	2	1	2
46	meena	1	19	2	1	2	1500	5	1	2	1	2	1	1	12.3	5	1	1	2	1	2	2	2	2	2	2	2
47	prem	1	18	1	2	5	8000	4	1	4	1	1	1	2	19	75	2	2	5	2	2	2	1	2	2	1	1
48	keran	4	54	1	1	1	900	5	1	2	2	1	1	2	2	180	3	2	5	1	2	1	1	2	2	1	1
49	kavya	4	55	2	5	9	9	6	1	2	1	1	1	1	17.3	.	.	2	2	1	1	2	1	2	2	2	2
50	lakshmi	2	26	2	1	2	1200	9	2	10	1	1	1	1	10	.	.	2	6	1	1	1	1	2	2	2	1
51	reeja	2	25	2	1	1	75	4	1	2	1	2	1	1	12	.	.	2	12	1	1	2	1	2	2	2	2
52	sundaramm	4	50	2	1	1	600	4	1	1	1	1	1	2	19	.	.	2	2	1	2	1	1	2	2	2	2
53	manju	1	18	2	2	1	25	5	2	3	2	2	1	1	14	.	.	2	.	1	2	2	1	2	2	1	2

Sl no	breathlss	pulserate	pulserhy	SBP	DBP	sinusrhy	sinusbrad	sinustach	exitblk	atrialect	jnrhythm	AV diss	firstdeg	secdeg	third deg	st change	cardiotox	bld sug	bld urea	s creat	sodium	potassm	gastr lvg	inj atrp	orceprnlin	steroids	outcome
1	2	88	1	100	60	1	2	2	2	2	2	2	2	2	2	2	1	92	24	0.8	139	4	1	2	2	2	4
2	2	84	2	120	70	2	2	2	1	2	2	2	2	2	2	2	3	89	17	0.8	155	4.9	1	1	1	2	1
3	2	60	1	120	80	2	1	2	2	2	2	2	1	2	2	1	2	102	46	1.2	136	4.6	2	1	1	2	1
4	2	66	2	120	86	2	1	2	2	2	1	2	2	2	2	1	3	155	32	1.1	135	3.8	1	1	1	2	1
5	2	62	1	110	70	2	1	2	2	2	2	2	2	2	2	2	2	60	18	0.7	138	4	1	1	1	2	1
6	2	84	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	81	20	0.8	138	3.9	1	2	1	2	4
7	2	60	2	120	60	2	2	2	2	2	2	2	2	2	2	2	1	123	40	1	140	4.2	1	1	1	2	1
8	2	48	1	120	80	2	1	2	2	2	2	2	2	2	2	2	2	190	50	1	137	4.5	1	1	1	2	1
9	2	68	1	120	90	1	1	2	2	2	2	2	2	2	2	2	2	69	21	0.9	148	4.8	1	1	1	2	1
10	2	76	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	105	58	1.6	134	4.1	2	1	1	2	1
11	2	80	1	110	80	1	1	2	2	2	2	2	2	2	2	1	2	62	15	1.2	130	3.5	1	1	1	2	1
12	2	46	1	110	70	1	2	2	2	2	2	2	2	2	1	1	3	89	38	1	138	4.2	1	1	1	2	1
13	2	110	1	110	70	1	1	1	2	2	2	2	2	2	2	1	2	77	18	0.7	139	3.8	1	1	1	2	1
14	2	84	1	120	70	1	2	2	2	2	2	2	2	2	2	2	1	185	19	0.9	132	3.6	1	1	1	2	1
15	2	110	1	100	60	1	2	1	2	2	2	2	2	2	2	1	2	82	43	0.8	136	3.8	1	1	1	2	1
16	2	120	1	110	80	2	2	1	2	2	2	2	2	2	2	2	1	68	26	0.9	132	3.1	1	1	1	2	1
17	2	58	1	100	70	2	1	2	2	2	2	2	2	2	2	2	2	67	20	0.7	146	3.8	1	1	1	2	1
18	2	100	1	100	80	2	2	1	2	2	2	2	1	2	2	1	2	88	48	0.9	132	3.8	1	1	1	2	1
19	2	62	1	110	60	1	2	2	2	2	2	2	2	2	2	2	1	60	18	0.6	139	3.7	1	1	1	2	1
20	2	48	2	120	80	2	2	2	2	2	2	2	2	2	1	1	3	64	43	0.6	136	3.5	1	1	1	2	1
21	2	72	1	100	70	1	2	2	1	2	2	2	2	2	2	1	3	82	20	0.9	.	4.2	1	2	1	2	1
22	2	72	1	110	80	2	1	2	2	2	2	2	2	2	2	1	2	107	32	1.2	.	4.2	1	1	1	2	1
23	2	70	1	130	80	1	2	2	2	2	2	2	2	2	2	2	1	172	24	0.8	.	.	1	1	2	2	1
24	2	58	2	100	70	2	1	2	2	2	2	2	2	2	2	1	2	145	21	0.8	.	.	1	1	1	2	1
25	2	104	1	110	70	1	2	2	2	2	2	2	2	2	2	1	2	137	21	0.8	.	.	1	2	1	2	1
26	2	80	1	100	70	1	2	2	2	2	2	2	2	2	2	2	1	68	29	0.9	.	.	1	1	1	2	4
27	2	78	1	130	80	1	2	2	2	2	2	2	2	2	2	2	1	105	28	0.8	.	.	1	2	1	2	1
28	2	80	1	150	100	2	1	2	2	2	2	2	2	2	2	2	2	86	19	1	.	.	1	1	1	2	1
29	2	96	1	120	70	1	2	2	2	2	1	2	2	2	2	2	3	108	31	0.9	.	.	1	2	2	2	4
30	2	92	1	90	70	1	2	2	2	2	2	2	2	2	2	2	1	80	22	0.7	.	.	1	2	1	2	1
31	2	94	1	100	70	1	2	2	2	2	2	2	2	2	2	1	2	96	32	0.9	.	.	1	2	1	2	1
32	2	90	1	140	90	2	1	2	2	2	2	2	2	2	2	2	2	81	18	0.8	.	3.8	1	2	1	2	1
33	2	120	1	130	80	2	2	1	2	2	2	2	2	2	2	2	1	130	34	0.9	.	4.5	1	2	1	2	1
34	2	56	2	100	70	2	2	2	2	2	1	2	2	2	2	1	3	113	21	0.8	136	4.2	1	1	1	2	1
35	2	92	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	118	16	0.9	140	4.1	1	2	1	2	4
36	2	68	1	100	70	1	2	2	2	2	2	2	2	2	2	2	1	104	28	1	138	3.9	1	2	1	2	4
37	2	86	1	120	70	1	2	2	2	2	2	2	2	2	2	2	1	82	18	0.9	136	4	1	2	2	2	1
38	2	90	1	130	90	1	1	2	2	2	2	2	2	2	2	1	2	90	18	0.8	.	4.2	1	1	1	2	1
39	2	42	2	110	70	2	1	2	2	2	2	2	2	2	2	2	2	72	35	1	136	4.1	2	1	1	2	1
40	1	110	2	90	60	2	2	2	1	1	2	2	2	2	2	2	3	65	26	0.7	132	4.8	1	1	1	2	1
41	2	110	1	110	80	1	2	1	1	2	2	2	2	1	2	2	3	70	20	0.7	.	.	1	2	1	2	1
42	2	40	2	70	40	2	1	2	2	2	2	2	2	1	2	1	3	60	28	0.9	.	.	1	1	1	2	1
43	2	64	1	90	70	1	2	2	2	2	2	2	2	2	2	1	2	98	31	0.9	138	4.1	1	2	1	2	1
44	2	84	1	110	80	1	1	2	2	2	2	2	2	2	2	1	2	72	28	0.9	138	3.9	2	1	1	2	1
45	2	76	1	140	100	1	1	2	2	2	2	2	2	2	2	1	2	112	26	0.8	143	3.9	1	1	1	2	1
46	2	70	1	110	80	2	1	2	2	2	2	2	2	2	2	1	2	87	23	0.7	138	4.1	2	1	1	2	1
47	1	100	1	120	76	1	1	2	2	2	2	2	2	2	2	2	2	86	30	0.9	136	4	1	1	1	2	1
48	2	64	2	140	100	2	1	2	2	2	2	2	2	2	2	1	2	87	25	1	138	3.9	1	1	2	2	1
49	2	66	1	100	70	1	1	2	2	2	2	2	2	2	2	2	2	82	28	0.9	132	5.1	1	1	1	2	1
50	2	102	1	120	80	1	2	2	2	2	2	2	2	2	2	1	2	64	30	1.4	136	3.8	1	1	1	2	1
51	2	102	1	130	86	1	2	2	2	2	2	2	2	2	2	2	1	64	20	0.8	138	3.7	2	1	1	2	1
52	2	82	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	101	40	1.4	140	3.9	1	1	1	2	1
53	2	96	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	77	26	0.7	136	3.2	1	2	1	2	4

Sl no	name	age class	age	sex	marital st	incomed	income	hosp stay	part ing	quantity	methings	consump	intentn	time	timeingst	timefrsai	timeintrv	firstaid	trtm outs	vomiting	abdmnlp	diarrhoea	giddiness	numbnes	mentalst	vision	palptatn
54	arun	2	25	1	2	9	9	1	1	1	3	2	4	2	21	.	.	2	1	2	2	2	2	2	2	2	2
55	sujina	1	19	2	1	9	9	5	2	2	2	2	4	1	18	.	.	2	2	1	2	2	1	2	2	2	2
56	mariya	2	23	2	1	9	9	6	1	3	1	1	1	1	7	.	.	1	8	1	2	1	1	2	2	1	1
57	suresh	3	32	1	1	3	2000	5	1	4	2	2	1	1	17.3	75	2	2	3	1	2	1	2	2	2	1	2
58	megala	3	30	2	1	1	600	5	1	3	1	1	1	2	18.5	.	.	2	2	1	2	2	1	2	2	2	2
59	geeta	1	20	2	1	9	9	3	2	1	1	1	1	2	1.3	.	.	1	3	1	2	2	2	2	2	2	2
60	arun	2	23	1	1	3	2000	6	1	5	1	1	1	1	10	.	.	1	3	2	2	1	1	2	2	2	2
61	rani	2	28	2	2	1	800	4	2	1	1	1	1	1	10	.	.	2	2	1	2	2	2	2	2	2	1
62	shamila	1	18	2	2	1	600	6	2	2	2	1	1	1	8	.	.	2	2	1	2	1	2	2	2	1	1
63	ganeshan	3	36	1	1	5	30000	4	2	2	1	1	1	1	10.3	.	.	1	28	1	2	2	1	2	2	2	2
64	malliga	2	25	2	1	9	9	4	1	2	2	2	1	1	17	.	.	2	28	1	1	2	1	2	2	2	2
65	priya	4	40	2	1	1	500	5	2	3	1	1	1	1	14.5	.	.	1	9	1	2	2	2	2	2	2	1
66	shajina	1	20	2	3	9	9	5	2	5	2	1	1	1	14.5	.	.	2	28	1	2	1	2	2	2	1	2
67	reena	2	27	2	1	2	1500	6	2	6	1	1	1	1	8.3	.	.	2	3	1	2	2	1	2	2	1	1
68	vadivel	1	17	1	2	9	9	4	2	1	3	1	1	1	6	.	.	2	5	2	2	1	2	2	2	2	2
69	murugan	3	38	1	1	2	1500	4	2	3	2	1	1	1	15	.	.	2	2	1	2	2	1	2	2	2	2
70	suresh	2	27	1	1	5	12500	6	2	4	2	1	1	1	13	.	.	2	2	2	2	1	2	2	2	2	1
71	jose	2	25	1	2	5	4000	6	2	4	2	1	1	1	13	.	.	2	2	2	2	2	1	2	2	1	2
72	saranya	2	28	2	1	5	5000	6	1	4	1	1	1	1	6	.	.	2	3	1	2	1	2	2	2	2	2
73	anuj	1	15	1	2	9	9	5	2	5	2	2	2	1	13.3	.	.	2	3	2	2	2	1	2	2	1	1
74	arun	2	28	1	1	9	9	5	2	2	1	1	1	2	20.3	.	.	2	2	1	2	2	1	2	2	2	1
75	suresh	3	32	1	1	4	3000	5	2	2	1	1	1	1	17.3	.	.	2	3	1	2	2	1	2	2	2	1
76	saju	2	21	1	2	2	1600	1	1	1	3	1	4	1	7	.	.	1	2	2	2	2	2	2	2	2	2
77	nataraj	3	37	1	4	2	1500	5	2	2	2	1	1	1	16	.	.	1	1	2	2	2	1	2	2	2	2
78	geeta	1	20	2	4	9	9	4	2	4	2	1	1	1	10	.	.	1	5	1	2	1	1	2	2	2	2
79	saji	3	31	1	1	2	1800	1	2	5	1	2	1	1	14.3	.	.	2	4	1	1	2	1	2	2	2	1
80	rija	2	22	2	2	5	5000	5	2	5	1	1	1	1	17	.	.	1	.	1	1	1	1	2	2	2	2
81	rani	1	18	2	2	2	1500	5	2	3	2	1	4	1	17.3	.	.	1	3	2	2	2	2	2	2	2	2
82	satheesh	2	23	1	2	2	1500	3	2	2	3	1	4	1	14.3	.	.	2	15	2	2	2	2	2	2	2	2
83	chinnan	2	25	1	1	9	9	3	1	3	2	1	4	1	7.3	.	.	2	2	2	2	2	2	2	2	2	2
84	murugan	2	23	1	2	3	2000	8	2	4	2	1	1	1	9	.	.	2	62	1	2	2	1	2	2	2	1
85	karthik	1	18	1	2	1	300	4	1	3	1	1	1	1	18	.	.	2	62	1	2	2	2	2	2	2	2
86	manju	1	16	2	2	2	1200	5	1	0	1	2	2	1	9.3	.	.	2	3	1	2	2	2	2	2	2	2
87	seeja	1	16	2	2	2	1500	5	1	1	1	2	1	1	9	.	.	2	7	1	1	2	1	2	2	2	2
88	nataraj	4	50	1	1	1	600	5	1	2	2	1	1	1	9	.	.	2	4	2	2	2	1	2	2	1	2
89	muthamma	4	62	2	5	1	900	7	1	1	2	1	1	1	7	.	.	2	4	1	2	2	1	2	2	2	1
90	reena	1	19	1	2	1	1000	4	1	2	2	2	1	1	9	.	.	1	6	1	2	1	1	2	2	2	2
91	sajinaa	2	27	1	2	1	1000	5	1	1	2	2	1	1	12	.	.	1	2	1	1	2	1	2	2	2	1
92	rani	2	25	2	2	5	4000	8	1	1	2	1	1	2	22	.	.	1	2	1	2	2	2	2	2	2	1
93	satheesh	2	27	1	1	5	5000	6	1	2	1	1	1	1	17.3	.	.	2	3	2	2	2	1	2	2	2	2
94	geeta	3	30	2	1	9	9	5	1	2	1	1	1	1	9	.	.	2	8	1	2	2	1	2	2	2	1
95	saranya	3	36	2	1	9	9	4	1	1	2	1	1	1	12	.	.	1	2	2	2	2	2	2	2	2	2
96	malliga	2	22	2	2	4	3000	4	1	2	2	2	1	1	13	.	.	2	11	2	2	2	2	2	2	2	2
97	lakshmi	2	23	2	2	1	750	5	1	2	1	1	1	1	14	.	.	2	5	1	1	2	1	2	2	2	1
98	rani	1	18	2	2	2	1800	4	1	1	1	1	1	1	14	.	.	2	3	1	2	1	2	2	2	1	2
99	nataraj	3	35	1	3	9	9	6	2	1	1	2	1	1	15	.	.	1	7	1	1	2	2	2	2	2	2
100	sumesh	2	21	1	2	4	3000	5	1	1	1	2	1	1	10	.	.	1	4	1	2	2	1	2	2	2	2
101	ponnamma	4	50	2	2	1	600	5	2	6	1	1	1	1	14	.	.	2	6	1	1	1	1	2	2	2	1
102	saji	3	35	1	1	3	2800	5	2	4	1	1	1	1	15	.	.	2	2	1	2	1	2	2	2	2	2
103	reena	1	13	2	2	9	9	4	2	2	1	2	1	1	16	10	1	1	4	2	2	2	2	2	2	2	2
104	shajila	1	18	2	2	1	800	6	2	5	1	2	1	1	10	390	3	1	9	2	2	2	1	2	2	1	2
105	geeta	2	28	2	1	9	9	2	1	1	3	1	4	1	7.3	45	1	1	2	2	2	2	2	2	2	2	2
106	meena	2	27	2	1	1	900	1	2	18	2	2	1	1	17.3	.	.	2	1	1	2	2	1	2	2	2	1

Sl no	breathlss	pulserate	pulserhy	SBP	DBP	sinusrhy	sinusbrad	sinustach	exitblk	atrialect	jnrhythm	AV diss	firstdeg	secdeg	thrd deg	st change	cardiotox	bld sug	bld urea	s creat	sodium	potassm	gastr lvg	inj atrp	orceprnln	steroids	outcome
54	2	92	1	120	70	1	2	2	2	2	2	2	2	2	2	2	1	104	18	0.8	140	4.1	1	2	2	2	1
55	2	86	1	100	70	1	2	2	2	2	2	2	2	2	2	2	1	69	16	0.7	139	4.2	1	2	1	2	1
56	2	100	1	90	60	2	1	1	2	2	2	2	2	2	1	2	3	79	27	0.9	129	4.4	2	1	1	2	1
57	2	108	1	120	70	1	1	1	2	2	2	2	2	2	1	2	3	132	22	0.8	137	3.6	2	1	1	2	1
58	2	110	1	110	70	2	1	1	2	2	2	2	2	2	2	1	2	88	28	1	137	4.7	1	1	1	2	1
59	2	86	1	120	70	1	2	2	2	2	2	2	2	2	2	1	2	60	20	0.7	149	5.6	1	1	1	2	1
60	2	80	1	110	80	1	1	2	2	2	2	2	2	2	2	1	2	124	26	0.9	143	3.9	1	1	1	2	1
61	2	86	1	120	70	1	2	2	2	2	2	2	2	2	2	1	2	61	27	0.8	160	4.1	1	1	1	2	4
62	1	90	1	110	80	2	2	2	2	2	2	2	2	2	1	1	3	85	36	1	138	4.2	1	1	1	2	1
63	2	100	1	110	70	1	2	2	2	2	2	2	2	2	2	1	2	130	20	0.9	143	3.6	1	2	1	2	1
64	2	64	1	120	70	1	2	2	2	2	2	2	2	2	2	2	1	73	18	0.6	132	3	2	1	1	2	1
65	1	80	1	150	70	1	2	2	2	2	2	2	2	2	2	1	2	120	26	0.9	142	4	1	1	1	2	1
66	2	70	1	120	70	1	1	2	2	2	2	2	2	2	2	1	2	76	18	0.8	138	3.5	1	1	1	2	1
67	2	88	2	130	70	2	2	2	1	2	2	2	2	2	2	1	3	62	24	1	143	4	1	1	1	2	1
68	2	94	1	110	80	1	2	2	2	2	2	2	2	2	2	2	1	77	25	0.8	133	3.7	1	2	1	2	1
69	2	60	1	120	90	1	2	2	2	2	2	2	2	2	2	2	1	104	28	0.8	140	4	1	2	1	2	1
70	2	84	1	110	70	2	2	2	1	2	2	2	2	2	2	1	3	157	5	0.9	138	4.4	1	1	1	2	1
71	2	84	1	100	70	1	2	2	2	2	2	2	1	2	2	2	2	110	22	0.6	145	3.9	1	1	1	2	1
72	2	60	1	100	70	2	2	2	2	2	2	2	1	2	2	1	2	121	26	1.2	138	4	1	1	2	2	1
73	2	96	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	88	28	0.6	136	4.6	1	2	1	2	1
74	2	88	1	120	70	1	2	2	2	2	2	2	2	2	2	2	1	92	30	1	146	4	1	2	1	2	1
75	2	58	1	110	60	2	1	2	2	2	2	2	2	2	2	2	2	62	44	1	142	4.2	1	1	1	2	1
76	2	86	1	100	80	1	2	2	2	2	2	2	2	2	2	2	1	84	18	0.8	138	4.1	1	2	2	2	4
77	1	92	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	103	20	0.9	136	4	1	1	1	2	1
78	2	74	1	110	70	1	2	2	2	2	2	2	2	2	2	1	2	60	37	1	130	4.8	1	1	1	2	1
79	2	36	1	90	80	2	2	2	2	2	2	1	2	2	2	2	3	.	.	.	.	.	2	1	1	2	2
80	2	52	2	100	60	2	2	2	2	2	2	2	2	2	2	2	.	130	21	0.8	139	4	1	1	1	2	1
81	2	86	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	60	28	0.9	138	4	1	2	1	2	1
82	2	66	1	120	80	2	1	2	2	2	2	2	2	2	2	2	2	74	16	0.7	137	4	1	1	1	2	4
83	2	96	1	110	80	1	2	2	2	2	2	2	2	2	2	2	1	75	16	0.8	122	3.2	1	1	1	2	1
84	1	80	2	110	70	2	1	2	2	2	2	1	1	2	2	1	3	80	23	0.8	140	3.8	1	1	1	2	1
85	2	56	1	110	80	1	2	2	2	2	2	2	2	2	2	2	1	79	42	0.9	136	3.9	1	1	2	2	1
86	2	100	1	100	80	1	2	2	2	2	2	2	2	2	2	1	2	78	40	0.8	145	4	1	1	1	2	1
87	2	80	1	100	70	1	2	2	2	2	2	2	2	2	2	2	1	75	24	0.9	138	3.4	1	1	1	2	1
88	2	42	1	100	70	2	2	2	2	2	2	2	2	2	1	2	3	88	24	1	136	4	1	1	1	2	1
89	2	120	1	110	70	2	1	1	2	2	2	2	2	2	2	1	2	77	18	0.8	142	4.1	1	1	1	2	1
90	2	68	1	110	60	1	2	2	2	2	2	2	2	2	2	2	1	88	34	0.9	140	3.6	1	2	1	2	4
91	2	60	1	110	70	2	1	2	2	2	2	2	2	2	2	2	2	67	37	1	138	3.6	1	1	1	2	1
92	2	60	2	110	90	2	2	2	1	2	2	2	2	2	2	1	3	58	16	0.7	136	3.5	1	1	1	2	1
93	1	68	1	130	70	1	1	2	2	2	2	2	2	2	2	2	2	82	34	0.9	139	4	1	1	1	2	1
94	1	80	1	110	90	1	1	2	2	2	2	2	2	2	2	1	2	62	33	0.7	142	4	1	1	2	2	1
95	2	96	1	120	60	1	2	2	2	2	2	2	2	2	2	2	1	72	30	0.9	144	3.5	1	1	1	2	1
96	2	80	1	130	80	1	2	2	2	2	2	2	2	2	2	2	1	110	49	0.8	138	3.4	1	2	2	2	1
97	2	50	1	90	80	2	1	2	2	2	2	2	2	2	1	2	3	120	31	1	148	4.5	1	1	1	2	1
98	2	60	2	100	70	2	2	2	2	2	2	2	1	2	2	1	2	86	55	1.3	140	4.5	1	1	1	2	1
99	2	98	1	110	70	1	2	2	2	2	2	2	2	2	2	2	1	126	25	0.8	144	4.5	1	2	1	2	1
100	2	92	1	120	70	1	2	2	2	2	2	2	2	2	2	2	1	67	21	0.8	130	4.3	1	2	1	2	1
101	2	88	1	150	90	1	2	2	2	2	2	2	2	2	2	1	2	235	34	1	137	4.3	1	1	1	2	1
102	2	54	1	140	80	2	1	2	2	2	2	2	1	2	2	1	2	62	16	0.9	138	3.6	1	1	1	2	1
103	2	140	1	100	70	2	2	1	2	2	2	2	2	2	2	2	1	80	25	0.6	132	4.2	1	1	1	2	1
104	2	120	1	110	70	2	1	1	2	2	2	2	2	2	2	1	2	128	72	1.8	140	3.4	1	1	1	2	1
105	2	92	1	110	80	1	2	2	2	2	2	2	2	2	2	2	1	88	28	0.8	141	4.1	1	2	2	2	4
106	2	48	2	70	40	2	2	2	2	2	2	2	2	2	2	2	2	.	.	.	.	.	2	1	2	2	2